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FRACTURES OF THE SPHENOIDAL SINUS WITH CEREBROSPINAL RHINORRHOEA

BY

WALPOLE LEWIN, M.S., F.R.C.S.

AND

Sir HUGH CAIRNS, K.B.E., D.M., F.R.C.S.

(From the Division of Surgery, Radcliffe Infirmary, Oxford, and the Military Hospital for Head Injuries, Wheatley)

It is now generally accepted that cerebrospinal fluid (C.S.F.) rhinorrhoea following a head injury indicates a dural tear in relation to a fracture involving the paranasal sinuses, and that a fascial repair of the torn dura is required as an insurance against intracranial infection in the future. Most of the fractures involve the frontal or ethmoid sinuses * In this paper we consider only the relatively uncommon group in which the rhinorrhoea was due to fracture of the sphenoidal sinus. Such fractures are not always detected by radiological examination, and a full exploration of this region involves special problems of exposure and accessibility. It seems that there are certain clinical characteristics which may lead to their detection pre-operatively.

Anatomy

The sphenoidal air sinuses lie in the body of the sphenoid bone and are usually asymmetrical and

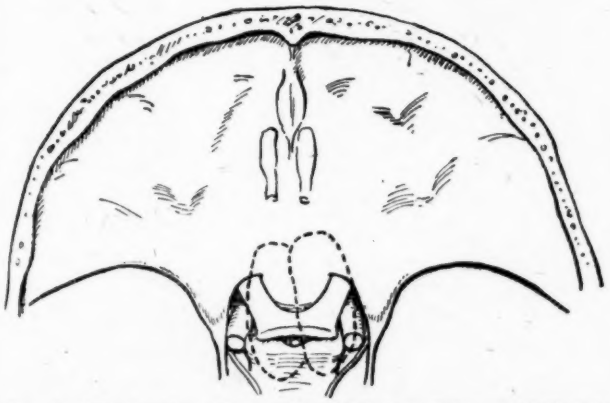

FIG. 1.—The outline of the sphenoidal sinus (interrupted line) in relation to the base of the skull.

separated from one another by a bony septum. They vary considerably in size and may extend into a well-marked lateral recess in the great wing of the sphenoid. Fig. 1, drawn from a necropsy specimen with the dura intact, shows that the sinus extends forward into the anterior fossa, and also laterally beneath the optic foramina. The sphenoid sinus may therefore be implicated by fractures running across the posterior part of the anterior fossa as well as by fractures of the middle fossa. The close relationship of the sinus to the optic nerves and chiasm, to the pituitary body, and laterally to the carotid arteries is apparent. And in front of the optic chiasm and the upper part of the subjacent pituitary body is the cisterna chiasmatica, which is continuous on each side with the arachnoid sheath of the optic nerves.

*In exceptional circumstances the fracture involves the petrous bone and the C.S.F. passes into the nose by way of the middle ear and Eustachian tube (Cairns and Lewin, to be published).

Incidence

In the years 1938–46, at the Radcliffe Infirmary, Oxford, and the Military Hospital for Head Injuries, Oxford, we have seen 84 patients with cerebrospinal rhinorrhoea following closed head injury. In 11 there was demonstrated at operation or necropsy a dural tear in relation to a fracture of the sphenoidal sinus. Six of these, however, must be excluded from the present examination because they also had other dural tears in relation to the frontal or ethmoidal sinuses, and it was therefore not possible to say with certainty which tear was responsible for the rhinorrhoea.

In the other five patients the dura was torn only over the sphenoidal sinus, and these cases are examined in closer detail. Three patients underwent a fascial repair and have remained well since, nearly four years after the operation. The other two patients died of pneumococcal meningitis, both in the days before penicillin therapy. One of these was admitted moribund and died within 24 hours of admission; the other developed meningitis on the day before operative repair was proposed, and he died nine days later.

The Fracture

The sphenoidal sinus may be fractured in several ways following head injury.

1. After a blow directed to the side of the head, the skull breaks in the coronal plane, either through the

coronal suture, which is splayed, or just behind the suture and anterior to the parietal eminences. The fracture runs down into each temporal fossa and then across the middle fossa to involve the wall of the sphenoidal sinus at the tuberculum sellae or in the anterior wall of the sella turcica (two cases).

2. A head-on blow produces a severe central frontal fracture with comminution of both walls of the frontal sinuses, from which fracture lines run backwards to involve the ethmoid and occasionally sphenoidal sinuses. In some of these cases there is a large central block fracture involving the whole paranasal sinus area of both sides (two cases).

3. The head-on blow may be taken lower down, so that the bony injury is predominantly facial, resulting in a central block fracture of the maxilla, and in some cases a fracture of the mandible as well. The overlying cribriform plate may be comminuted. This is a fracture which on routine radiography may seem to be entirely extracranial: the extent of the damage to the cribriform plate and sphenoidal sinus may not be fully appreciated unless special radiographic projections of the paranasal sinuses are taken through the orbit, as recommended by Davies (Calvert, 1942) and Johnson and Dutt (1947) (one case).

4. The above are the ways in which the fractures occurred in our series. In addition it is possible that there may be discrete fractures of the skull limited to the roof of the sphenoidal sinus, similar to those which have been seen on the roof of the ethmoidal cells at operation and necropsy (Johnson and Dutt, 1947; Cairns, 1949, Fig. 8).

The Dural Tear.—This may be extensive, but we have had to exclude from this series cases in which the gap in the dura involved the ethmoidal as well as the sphenoidal roof. In the five cases in which the dural tear was limited to the sphenoidal sinus area, the tear was usually only a few millimetres wide and was situated over the tuberculum sellae or the anterior wall of the sella turcica. It probably resulted from splintering of the thin bone in this region.

The Cerebrospinal Fluid Rhinorrhoea

The outstanding feature of these sphenoidal cases is the nature of the rhinorrhoea. It is characteristically profuse. In these five patients it was so described, and that it should have been so recorded by different clinicians renders what is an essentially qualitative observation the more reliable. In all patients the rhinorrhoea, once begun, continued unabated until operation or death, in one case for six months.

The following case record gives a typical description of the rhinorrhoea.

Case 1.—This patient sustained a coronal fracture of the skull at the level of the coronal suture following a motorcycle accident. C.S.F. rhinorrhoea was present from the time of injury, and a note made 15 days later reads: "This soldier continues to have profuse C.S.F. rhinorrhoea whenever he sits up and leans over. For the last few days it has been coming down the left nostril only, though hitherto it has sometimes been from the right and sometimes from the left nostril. When the patient is lying in the dorsal recumbent position he feels fluid passing down his nasopharynx and has to swallow from time to time. During the night, when he is asleep, it tends at times to get into his larynx and he is awakened with fits of coughing, sometimes severe. The patient was sat up this morning, and within a few moments watery fluid began to drip from the left nostril—about a drop every second or less. I collected 4 ml. for examination and then let him lie down. I think the drip would have continued indefinitely in the sitting position.

FIG. 2.—Case 2. Radiograph taken 26 days after the accident, showing air in cisterna chiasmatica, anterior horn of lateral ventricle, and subdural space.

In one patient (Case 2) a profuse rhinorrhoea began one week after the injury, at the same time as pneumococcal meningitis. The meningitis was treated with intrathecal penicillin, but showed a marked tendency to relapse. The C.S.F. escaping from the nose contained a considerable amount of penicillin, and it is not unlikely that the two relapses of meningitis, which occurred at two and four weeks after the accident, were associated with inadequate concentrations of penicillin within the meninges owing to its continual escape with C.S.F. from the nose. We have seen a similar "leakage" of

penicillin from the subarachnoid into the subdural space in an infant after head injury (Smith *et al.*, 1946, Case 8).

How common is such profuse rhinorrhoea? In our 79 cases of cerebrospinal fluid rhinorrhoea not due to sphenoidal sinus injury the rhinorrhoea was profuse in only four cases. These patients had large tears in relation to the frontal or ethmoidal sinus. The explanation of the high incidence of profuse rhinorrhoea in sphenoidal sinus tears must be anatomical and due to the fact that the dural tear establishes a communication between the cisterna chiasmatica and the nose. By contrast, the subarachnoid spaces over the frontal pole are small, so that profuse rhinorrhoea from tears further forward is correspondingly uncommon.

The time of onset of the rhinorrhoea after injury in these five patients was variable. It came on at once in two cases, but was delayed seven to ten days in two cases and nine weeks in the remaining case. Such variability is seen in all groups of traumatic rhinorrhoea. The onset of rhinorrhoea depends not only on the type of fracture but also on whether the dural tear is temporarily sealed by blood clot or brain tissue. For this reason rhinorrhoea after anterior fossa fractures may begin seven to ten days after injury, when the oedema of the surrounding brain tissue is subsiding, the pulped brain is liquefied, and the blood clot is undergoing organization. Other factors are necessary to explain the very delayed onset which is sometimes met with.

Aerocele.—With such a ready access to the basal cisterns, air may enter the cranium and be seen on *x*-ray examination of the skull. In one case, for the first month after injury air was seen in the subdural space, ventricle, and cisterna chiasmatica, but not in the brain. Fig. 2 shows the air in this cistern, and such localization should suggest that the dural tear is in relation to the sphenoidal sinus.

Other Signs

Preservation of Smell.—Theoretically, if the bony damage is confined to the sphenoidal sinus region and does not extend into the ethmoids, the sense of smell should be preserved, and this was in fact so in two of the four patients whom we have had under investigation. Nevertheless, no absolute reliance can be placed on this observation, for not only may severe frontal sinus fractures occur without disturbance of smell, but in apparently isolated sphenoidal cases the olfactory filaments may be damaged extracranially, particularly when the brunt of the injury is borne facially.

Injury to Neighbouring Structures.—Clinical evidence of damage to the hypothalamus and pituitary gland (diabetes insipidus, inverted sleep rhythm, glycosuria, or obesity), to the optic chiasm, or to the carotid artery was present in two of the five cases. One patient had diabetes insipidus; the other had a visual field defect due to damage to the optic chiasm and also a carotid aneurysm. The accompanying Table, compiled from our 200 cases of paranasal sinus fracture following closed head injury, shows that, though these neighbourhood signs are commoner in the presence of a sphenoid fracture, they are by no means rare with fractures involving the other paranasal sinuses.

Paranasal Sinus Fractures in Closed Head Injury (200 Cases). Incidence of Damage to Parasella Structures

Paranasal Sinus Fracture	No. of Cases	Hypothalamic Syndromes	Chiasmal Injury	Carotid Aneurysm or Fistula	Cases
Without fracture of the sphenoid	184	7	7	3	14
With fracture of the sphenoid	16	5	3	1	6
Total ..	200	12	10	4	20*

* Six patients had a combination of these symptoms.

Risk of Intracranial Infection

Three of our five patients developed meningitis, in all instances pneumococcal, two within a week of injury and the other eight months later. Two of these cases occurred before penicillin was available, and were fatal. The third patient was treated with penicillin, recovered from the meningitis, and subsequently underwent a successful fascial repair of his dura (Case 2).

In our total series of 84 cases of cerebrospinal rhinorrhoea the incidence of intracranial infection was 19%. Thus it would seem that the risk of meningitis is greater with sphenoidal sinus fractures than with fractures of the other paranasal sinuses.

The close proximity of the basal cisterns renders patients with sphenoidal sinus fracture particularly liable to meningitis, and dural repair should be done as soon as possible. Another reason for early operation is that if meningitis supervenes and the rhinorrhoea is profuse, it may be difficult to establish an adequate concentration of penicillin in the C.S.F. by intrathecal injections.

Diagnosis and Operative Treatment

The difficulties of obtaining a satisfactory repair of a dural tear over the sphenoidal sinus may be considerable, but they can be overcome if beforehand the possibility that the tear is over the sphenoidal sinus has been suspected and the operation has been planned to provide a good exposure of the sella turcica region. Radiographic demonstration of a fracture of the sphenoidal sinus may be extremely difficult, but this lesion should be suspected when, in the absence of radiological evidence of a gross fracture of the posterior wall of the frontal sinus or of the roof of the ethmoid, there is profuse rhinorrhoea. Additional evidence in favour of the sphenoidal location of the trauma is the presence of air in the cisterna chiasmatica, as shown by radiographs.

Faced with the possibility that the C.S.F. is escaping through the sphenoidal sinus region, the surgeon must be prepared to make a full bifrontal exposure and, if need be, to divide olfactory tracts which are functioning normally.

The intradural approach for dural repair, as used by Julian Taylor (Eden, 1942), is the only way by which these sphenoidal tears can be fully demonstrated. A bifrontal flap is turned. The dura is then opened along the anterior limb of the flap on each side of the sagittal sinus, and the inner surface of the dura covering the frontal and ethmoid sinuses is inspected for tears, first on one side and then on the other. If no gross tear, sufficient to account for the profuse rhinorrhoea, is found, the anterior fossa must be widely exposed. The anterior end of the sagittal sinus, the falx, and one or both olfactory tracts must be divided. It is then possible to retract both frontal lobes freely and thus to obtain a good view of the optic groove and sella turcica (Fig. 3).

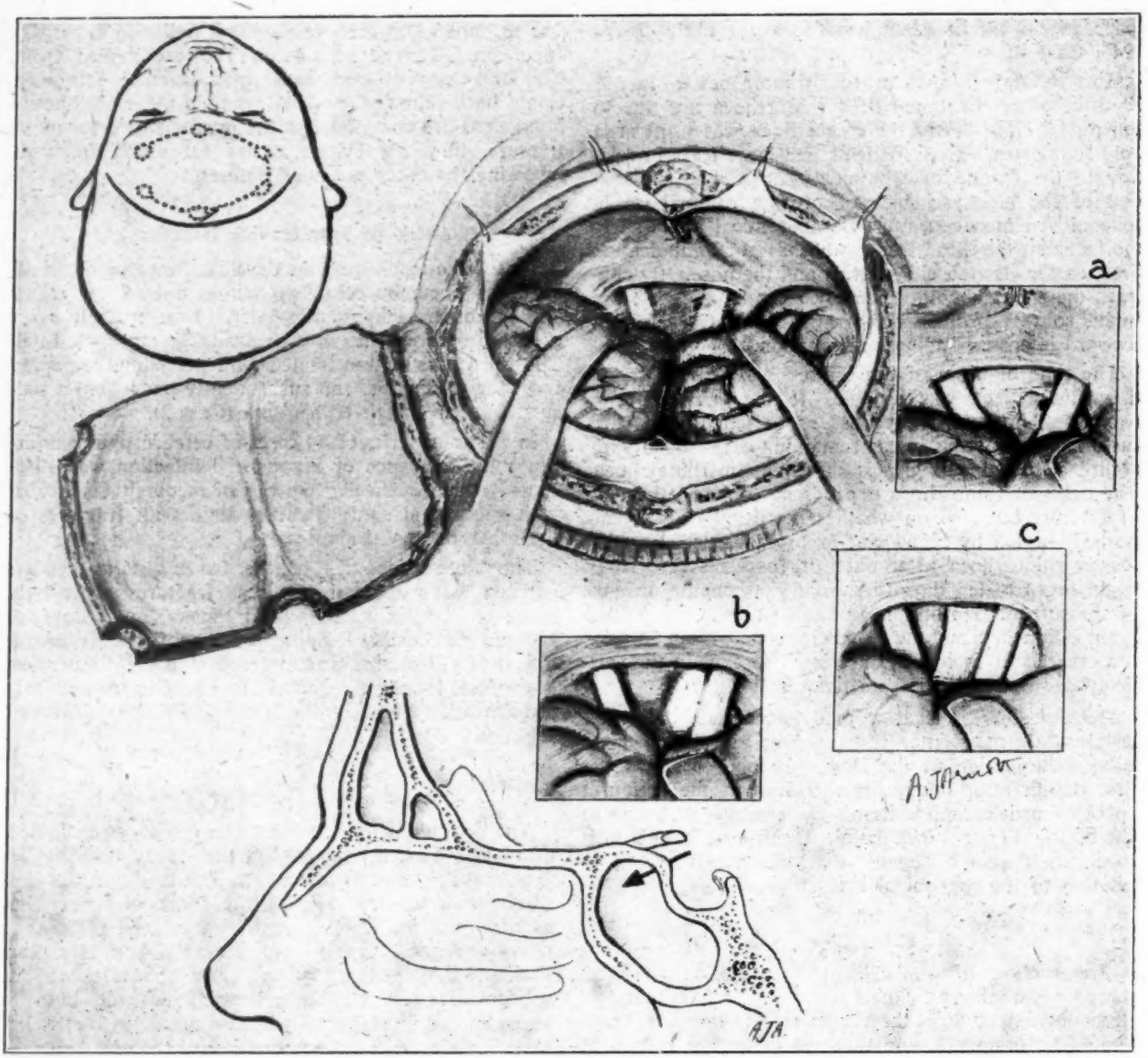

FIG. 3.—Case 2. Operation sketch. Bifrontal flap, division of falx and sagittal sinus, retraction of both frontal lobes. (*a*) Hole in anterior wall of sella visible after removal of arachnoid of cisterna chiasmatica. (*b*) Hole plugged with fibrin foam. (*c*) Patch of fascia lata applied

For full inspection of the anterior wall of the sella it is necessary to open widely the arachnoid of the cisterna chiasmatica.

The dural tear may be quite small and easily missed, and the proximity of the optic nerves and chiasm, and of the carotid artery, adds to the difficulty of the exploration and of the repair. In two of the three cases in this series that were repaired, the tear was found in the anterior wall of the pituitary fossa beneath one of the optic nerves; in the third case it lay over the tuberculum sellae. In each case the hole in the dura was repaired with a stamp of fascia lata which lay snug and was allowed to adhere by natural processes. We prefer fascia lata to temporal fascia because it is uniform in thickness, more pliable, and more likely to remain and adhere where it has been placed.

This method effectively stopped the rhinorrhoea in our three cases. Four years after the operation all were well and working; there had been no recurrence of rhinorrhoea and no post-operative meningitis.

The main features of the operation are illustrated by the following case.

Case 2

An officer aged 32 was involved in a car accident in Germany on May 10, 1945, and sustained severe facial injuries, including corrosive burns of both eyes, a central block fracture of the maxilla, fracture of the nose and of the right malar and left petrous bones, and fracture of the 6th thoracic vertebra. He also had paralysis of the left sixth and seventh cranial nerves. He bled from both nostrils, and for some hours there was a profuse discharge of blood-stained C.S.F. from his left ear. Smell was grossly impaired at each nostril.

The facial fractures were splinted, after a prophylactic intrathecal injection of 20,000 units of penicillin. Six days later the patient developed profuse bilateral rhinorrhoea and pneumococcal meningitis. He was treated with systemic and intrathecal penicillin and with sulphadiazine, and, after two relapses of his meningitis, finally improved.

The only skull fracture seen on *x*-ray examination was a fissure in the sella turcica. Several radiographs taken during

the first month after injury showed intracranial air at various times in the subdural space, ventricle, and the cisterna chiasmatica (Fig. 2). The significance of this latter finding has already been mentioned.

Operation to repair the dural tear and stop the rhinorrhoea could not be undertaken for some weeks. The patient was flown to Oxford from Germany two weeks after the accident, and at that time he had a relapse of his meningitis, and was developing collapse of his right lung with bronchopneumonia, which persisted for some weeks. Whenever he was turned on one or other side C.S.F. escaped from one or both nostrils, and it was easy to collect samples for penicillin studies at two-hourly intervals.

By June 22 his general condition was satisfactory for operation and his C.S.F. normal, though the rhinorrhoea was still present. Because radiographs suggested a fracture into the sphenoidal sinus and had also shown air in the cisterna chiasmatica, a wide exploration was carried out, after a preliminary lumbar intrathecal injection of 10,000 units of penicillin. A bifrontal bone flap was turned to the left. The anterior fossa was explored intradurally, and the dura covering the frontal sinuses, orbital roofs, and the ethmoidal regions looked quite normal. The olfactory bulbs and tracts also looked normal, and, though at this stage of his illness smell had partly recovered on the right side, it was clearly necessary to sacrifice them, divide the anterior extremity of the sagittal sinus and the falx cerebri, and retract both frontal lobes in order to examine thoroughly the sellar region (Fig. 3). At first nothing abnormal could be seen, but when the arachnoid of the cisterna chiasmatica was separated from the anterior wall of the sella an area of bare white bone (0.4 cm. in diameter) was disclosed on the right side below the optic groove and above the diaphragma sellae. In the right edge of this bare area, under the medial edge of the right optic nerve, was a hole through which passed a sleeve of arachnoid containing abundant C.S.F. When the arachnoid was cleared a probe could be passed through this hole into the sphenoidal sinus.

The bony hole was plugged with fibrin foam and then covered with a stamp of fascia lata. A second patch of fibrin foam was placed on the anterior wall of the sella between the right optic nerve and internal carotid artery (Fig. 3). The falx was then stitched back into place, and the dura, bone, and scalp were repaired in the usual way.

Subsequent progress was uneventful. The rhinorrhoea ceased immediately and did not recur. The maxillary fracture, which had been treated by internal splinting, healed in good position. The left facial palsy recovered spontaneously and completely, but there remained considerable deafness of the left ear and a scarred but intact ear drum. The spinal fracture, which was treated by a spinal jacket, consolidated with a wedge deformity. His left external rectus made no recovery, and subsequent operations on his extraocular muscles in order to banish the diplopia were only partly successful.

When seen four years after the accident the patient was well and fully employed in administrative work.

In this case the dural tear was limited to the anterior wall of the sella, and, so far as we were able to ascertain, so was the fracture. The rhinorrhoea did not come on until six days after the accident, and was accompanied by pneumococcal meningitis, which relapsed on two occasions in spite of energetic treatment with penicillin and sulphonamides. Much C.S.F. escaped from the nostrils, and with it penicillin. The patient's general condition did not permit of operation until six weeks after the accident, because of his jaw fractures, meningitis, and bronchopneumonia. Anything short of an extensive bilateral intradural exploration of the anterior fossa would have failed to reveal the dural tear, and this operation entailed sacrifice of his already partly damaged sense of smell. The indications for such an extensive operation were the profuse character of the rhinorrhoea in the absence of radiographic signs of gross fracture of the frontal or ethmoidal sinuses, and the presence of air in the cisterna chiasmatica. The final result may be regarded as highly satisfactory in view of the extensive and severe nature of his injuries.

Tears in the dura may be multiple, hence the surgeon must always satisfy himself that the tear which he finds is the one which is responsible for the rhinorrhoea and that a dural defect further back is not being overlooked. Such a possibility at operation can be envisaged by studying the following case record

Case 3

In 1938 a youth aged 18 was involved in a motor-cycle accident and sustained a compound depressed right fronto-parietal fracture, comminuting both walls of the frontal sinus and the anterior ethmoids on the right side. Special orbital views were not taken, and from the routine views there was no suspicion that the sphenoidal sinus was involved.

The fracture was elevated at another hospital on the day of the accident. Cerebrospinal fluid rhinorrhoea was observed from the *right* nostril on the day after the accident, and it continued for five weeks and then stopped. But a month later profuse rhinorrhoea began from the *left* nostril and continued for six months. The patient was admitted to the Radcliffe Infirmary and exploration was advised, but the day before operation the patient developed pneumococcal meningitis, from which he died 10 days later.

At necropsy, in addition to the signs of meningitis, two distinct fractures were demonstrated. One linear fracture ran down the back of the right frontal sinus, where part of the bone was missing and the dura was adherent to the mucosa ; but there was no actual tear of the dura. A second fracture ran across the left optic groove, and here the dura was torn and a hole 0.4 cm. in diameter led into the sphenoidal sinus.

We may assume that the rhinorrhoea from the right nostril followed the fracture behind the right frontal sinus, and healing finally occurred by the dura and sinus mucosa becoming adherent. The left rhinorrhoea was due to the sphenoidal sinus fracture. Owing to septal variations and injury, rhinorrhoea from one side may on occasion result from a contralateral dural tear. It would have been easy to miss this sphenoidal tear with operative exploration limited to the side of the known fracture. We do not advocate bilateral exploration in all cases of traumatic rhinorrhoea. There are some cases of frontal and ethmoidal fracture in which the clinical and radiographic signs provide clear-cut evidence of a unilateral lesion. But whenever there is suspicion that the leakage is through the sphenoidal sinus the exploration should be bilateral

Summary

In a series of 84 cases of C.S.F. rhinorrhoea following closed head injury, a dural tear over the sphenoidal sinus was found in 11 (13%) and was the only tear in relation to the paranasal sinuses in 5 (6%).

The main clinical characteristic of these five cases was the profuseness of the rhinorrhoea, due to the fact that the tear involved the overlying cistern chiasmatica Another sign which may be helpful in diagnosis is the radiographic demonstration of air in the cisterna chiasmatica, as in one of these cases.

Neighbourhood signs, such as evidence of damage to the hypothalamic region, optic chiasm, or carotid artery, were present in nearly half the sphenoid cases, but by themselves they do not necessarily indicate a fracture involving the sphenoid sinus. The preservation or loss of sense of smell is likewise no clinical guide.

Operative repair is urgently indicated. The risk of spread of infection intracranially into the basal cisterns is appreciably higher than with fractures involving the other paranasal sinuses.

Operative repair is difficult and requires a bifrontal flap, usually with division of the falx and sacrifice of one or both olfactory tracts. Three patients underwent successful dural repair and have since been followed for nearly four years. There has been no further rhinorrhoea or intracranial infection, and all are well and working.

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ABSENCE OF THE VASA AS A CAUSE OF AZOOSPERMIA

BY

KENNETH WALKER, F.R.C.S.

Emeritus Surgeon, Royal Northern Hospital; Consulting Urologist, Dartford Hospitals

AND

REYNOLD BOYD, F.R.C.S.Ed.

Director, Sterility Clinics, St. John's Hospital, Chelmsford, and Wanstead Hospital

The treatment of azoospermia due to a blockage in the system of efferent ducts by which the spermatozoa reach the posterior urethra is admittedly very unsatisfactory. The commonest situation for such a blockage is the epididymal canal, and when we consider the small calibre of this canal, its length, and the prevalence of epididymitis, the frequency with which it becomes occluded can easily be understood. The next commonest site for an obstruction is in the ejaculatory ducts, and this is generally the result of recurrent attacks of prostatitis and vesiculitis. Although the vas deferens may also be involved in the infection which brings about an epididymitis, a post-inflammatory stricture of it is uncommon. Genital tuberculosis is an exception to this generalization. In this disease the lumen of the vas is often obliterated, but as the epididymis is still more seriously affected this is of no practical importance. When the vas is obstructed it is usually from trauma, and the commonest cause is damage inflicted on it during the course of an operation such as herniotomy.

To diagnose the exact site of the blockage is not always an easy task. Should there be a clear history of bilateral epididymitis and additional evidence in the shape of areas of induration at the lower poles of both epididymides, then it may be assumed with confidence that the blockage is in these organs. But if there is no such history, and if no areas of induration are found on palpating the epididymides, then it is quite possible that the ejaculatory canals are at fault.

How is this supposition to be confirmed or refuted? Textbooks suggest that catheterization of the ejaculatory ducts should be carried out for this purpose. But unfortunately this advice is much easier to give than to follow. The openings of the ducts on the slopes of the verumontanum may be so placed that even when clearly seen it is mechanically impossible to pass a filiform bougie along them. One of us (K. W.) remembers witnessing an expert at catheterization at work and was not impressed with the results of his skill. A probe was forced into the lumen of the ducts through a direct-vision posterior urethroscope employing air distension, but such a manœuvre as this was far more likely to produce a stricture than dilatation. Trauma of the ducts must be rigorously avoided.

Another method of diagnosing an obstruction in an ejaculatory duct is to expose the vas at the neck of the scrotum and to inject into it a solution of indigo-carmine, noting afterwards whether this has passed into the posterior urethra or not. Some urologists have recommended injection of the vasa also, as a therapeutic measure. They believe that the ejaculatory ducts can be opened up thereby, just as the Fallopian tubes are opened up by insufflation. We have never seen a blockage removed in this way, and dislike puncturing vasa purely for diagnostic reasons, not only because of the damage inflicted on a delicate structure but also because of the risk of provoking a descending epididymitis.

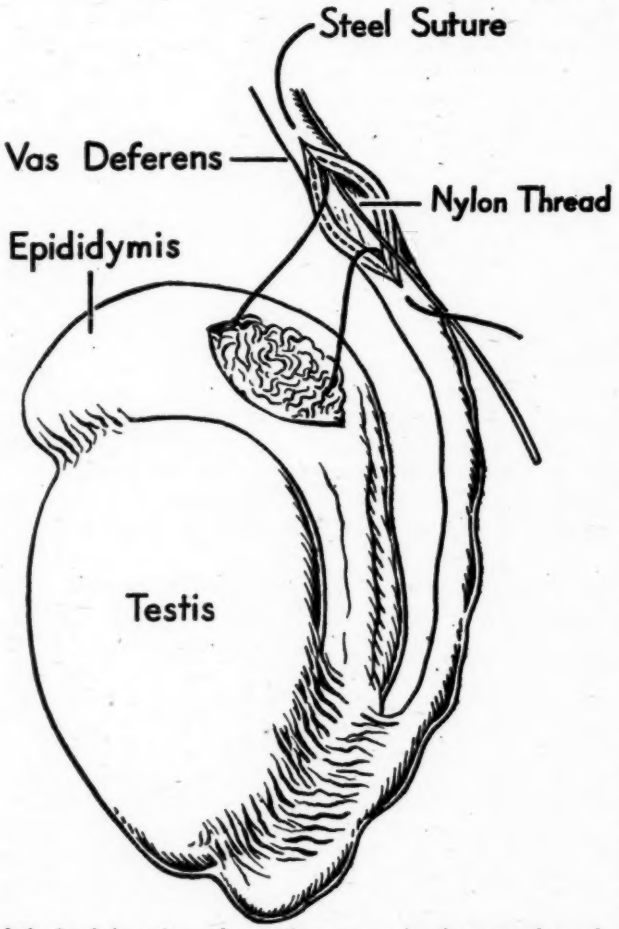
If a case of azoospermia is due to blockage of the epididymal canals, the only possible remedy is to carry out a vaso-epididymostomy. Although the results of this operation are very discouraging, we cannot afford to discard a method which in 10% of cases leads to the birth of a child and in 25% to the reappearance of spermatozoa in the semen. If the patient is prepared to accept the gamble and a biopsy has revealed active spermatogenesis in the testes, this operation is justifiable. It can do no harm even if it does no good. Some patients on whom we have operated had previously heard that an alternative method of treatment was available. This was to puncture the testes, withdraw spermatozoa from them, and inseminate with the aspirated fluid. Such treatment is, for reasons into which we do not propose to go, utterly useless. It need not even be considered.

Vaso-epididymostomy

It is useful, but not essential, to give a month's preliminary treatment with gonadotropic hormone in order to stimulate spermatogenesis and to ensure that the epididymal tubes are filled with secretion at the time of operating. After the testicles have been delivered from the scrotum and carefully inspected, the vas deferens is separated from surrounding structures at the level of the head of the epididymis, but great care should be exercised not to imperil its blood supply. The vas is next incised and some 5 to 6 in. (12.5 to 15 cm.) of nylon thread passed down it to demonstrate its patency. This thread is left *in situ* to act as a guide, and attention is then directed to the epididymis. An oval area of the tunica covering the underlying distended tubules is removed, and when the tubules are incised a drop of whitish fluid exudes. This is smeared on to a sterilized coverslip and the presence of spermatozoa confirmed. The finest round-bodied straight needle, carrying 40-gauge steel wire, is passed through the wall of the vas at the lower end of the incision into it and then through the exposed epididymal tubules. Having taken a good bite of these, the needle passes through the vas again, but at the upper end of the incision. It finally emerges through the skin of the scrotum near the root

of the penis (see illustration). When pulled upon, this wire invaginates the tubules into the lumen of the vas. During the subsequent healing it also acts as an excellent splint to the juncture between the vas and the epididymal canal. The lips of the vasal incision are finally united to the epididymis on each side by two lateral sutures of 0.005-in. tantalum wire. The operation is completed by returning the testicles into the scrotum and closing the latter, without drainage. The steel splinting wire is pulled out on the ninth day.

The real object of this paper is not so much to describe the details of the operation of vaso-epididymostomy as to call attention to anomalies of the vas which may be accidentally discovered in the course of performing it.



Method of insertion of splinting suture in the operation of vaso-epididymostomy.

In about 10% of the patients operated on by us either no vas at all was found, or else the vas was reduced to a thread-like structure devoid of any lumen. Theoretically, absence of the vasa might be due to one of two causes—congenital absence, or disappearance as the result of some previous disease. Reference to embryological and anatomical literature suggests that the former cause is an uncommon one.

That the second cause—disappearance through disease—is the more likely explanation is supported by what we found in three or four cases. At the level of the neck of the scrotum a vas was discovered, but on tracing it downwards it gradually narrowed, and 2 in. (5 cm.) before it should have joined the epididymis it disappeared altogether. It was noted that where it existed the vas was tortuous, solid, and very friable. On cutting into it a creamy fluid exuded, as though the vas was in a state of coagulative necrosis. Sometimes an opaque line marked the previous position of the vas in that part of its course from which it was absent. There was nothing in the patients' clinical history to throw light on the nature of the disease which had attacked the vasa and had led to their disappearance.

In two patients a somewhat similar condition was found in the epididymal canal. When seen through the tunica covering them the epididymal tubules had a light yellowish colour, and when cut into they were found to be solid. On pressure a fluid exuded, sometimes containing a few spermatozoa and sometimes without them. Again it looked as though some form of coagulation or fatty necrosis was in progress. In yet another two cases the vas ended abruptly 2 in. above the epididymis in a kind of corkscrew, recalling the terminal portion of the normal vas. Here, congenital malformation may well have been the explanation.

Conclusion

What is the moral to be drawn from these observations? The first and most obvious is that in all cases of obstructive azoospermia, and particularly if an operation is about to be undertaken to remedy it, the vasa must be very carefully palpated through the scrotum. Every effort must be exerted to make sure that they actually join the epididymides. Through neglect of this precaution one of our patients had been submitted to two years of useless hormone treatment by a specialist before he came to us for operation. It is true that in some cases it is difficult to trace the whole course of the vasa until they meet the epididymides, but if the preliminary examination had been more careful some of these exploratory operations might have been avoided. The second lesson to be learnt is that sterility is occasionally the result of some pathological condition of the vas the nature of which is still obscure. It is to be hoped that further research will throw light on this interesting problem.

The commercial production of adrenocorticotropic hormone in Britain has been in the stage of planning and organization for some time under the aegis of the National Research Development Corporation, the Government agency set up three years ago to undertake commercial exploitation of Government-owned patents and other technical projects. The three Government departments involved are the Ministry of Health, Medical Research Council, and Ministry of Food, which has organized the collection of some thousands of sheep, cow, and pig pituitary glands weekly from slaughterhouses up and down the country, solved the problem of their preservation, and delivered them to the five pharmaceutical firms who are co-operating in the attempt to produce A.C.T.H. to the required standards of purity and activity. One of these firms, Organon Ltd., has just delivered its first batch of product to the Ministry of Health, which is rigidly controlling distribution since supply is far short of demand, and likely to continue so unless the efficiency of the manufacturing process can be greatly raised or a synthetic substitute discovered. An annual production of 800 g. seems the probable maximum. There is no likelihood of cortisone being made in Britain, since its manufacture in about thirty complicated low-yield stages from ox-bile is covered by American patents, and in any case there is probably not enough British ox-bile available to start with. It will be recalled that the Ministry of Health recently began monthly distribution to 50 hospitals (24 teaching hospitals and 26 other hospitals nominated by regional boards) of 6 g. cortisone and 3 g. A.C.T.H. imported from the U.S.A. at about $50 a gramme.

SOME CONTRIBUTIONS OF MODERN GENETICS TO MEDICINE

BY

NORMAN T. J. BAILEY, M.A.

Statistician, Department of Medicine, University of Cambridge

Modern genetics stems from the work, published in 1866, of the Augustinian monk Gregor Mendel, who occupied his leisure by growing edible peas in the monastery garden. Mendel's work went almost unnoticed by his contemporaries, and it was not until 1900, after the ground had been prepared by the great advances in cytology during the 'eighties and 'nineties, that its importance was recognized more or less simultaneously by de Vries, Correns, and Tschermak. For just half a century now genetics has made rapid and constant progress and has found application to the whole range of living organisms, from viruses and bacteria to man himself. It is with the latter that medical genetics has hitherto been mainly concerned, though the former will probably occupy an increasingly important place in the subject.

It is the purpose of this article both to indicate the scope and importance of medical genetics and to emphasize the unique opportunities which exist for doctors to collect and use genetical data. The main fields of application will be covered, with particular reference to the practical use to be made of genetical theory. Special attention will be given to methods of recording data so as to ensure that the maximum amount of information can be extracted from them; and some of the special uses of family pedigrees will also be mentioned.

There is no need to do more than make a passing reference to the elementary principles on which practically the whole of genetics is based. Both of Mendel's laws may be summarized under the single heading of "particulate inheritance," according to which the basic units of heredity are material particles—the genes—which are transmitted unchanged from generation to generation. The first law, the law of segregation, states that characters are controlled by pairs of genes which segregate or separate from one another during the formation of germ-cells and pass into different gametes, which contain only half the full (diploid) quota of chromosomes and are therefore termed haploid. The pairs are restored at fertilization. Thus the genes are shuffled by segregation and redealt to the next generation. Characters controlled by genes may also segregate, and different types will appear amongst the offspring in certain definite proportions. The second law, the law of independent assortment, states that when two or more pairs of genes segregate simultaneously they do so independently of each other. This law has had to be modified by the existence of linkage. Briefly, chromosomes, and therefore genes in different chromosomes, are shuffled independently; but genes in the same chromosome, in which they are arranged in a linear order, tend to be handed on together, segregating and recombining, however, to an extent which depends on their distance apart.

Owing to the random element introduced by the shuffling process the analysis of genetical data must inevitably appeal to the ideas of uncertainty and probability associated, for example, with poker or bridge. With experimental plant or animal breeding the element of uncertainty can often be considerably reduced by using large progenies. In man, on the other hand, not only are families small but it is often difficult to find marriages between individuals of the appropriate genetic constitution. However, special methods are available for dealing with these difficulties, and a surprisingly large amount of useful information can often be extracted from apparently unpromising data.

There is another fundamental genetic phenomenon which must be mentioned; that is, the occurrence of mutations. Mutation is perhaps most satisfactorily defined as the beginning of a heritable variation. It can be applied to either genes or chromosomes. A gene may suddenly, though with extreme rarity, undergo a spontaneous change to a different form, and this change may be reflected in the characters which the gene controls. Again, chromosomes may be present in abnormal numbers, or a piece of one may break off and either be lost (deletion), or become reattached the wrong way round (inversion) or even joined on to a different chromosome (translocation). The word "mutation" should be applied only to the act of change in a hereditary unit. New genes which suddenly appear through mutation are called "mutant genes" and the characters to which they give rise "mutant characters."

Much of the early work on genetics was carried out by T. H. Morgan and his school, who bred the fruit-fly *Drosophila melanogaster* literally by the million. Though these studies may seem somewhat remote from medicine, the hereditary mechanisms thus elucidated have in fact proved to be of universal application.

Contributions to Plant and Animal Breeding

Substantial contributions have been made by genetics towards increasing the production of food, particularly in the cereal group. Considerable increases in the yield of maize were obtained by using breeding techniques designed to produce new varieties of "hybrid corn." Again, the manufacture of textiles has been aided by producing cotton plants with longer and stronger fibres, and by breeding sheep with heavier and finer fleeces. Useful work has also been done on the resistance of plants and animals to pests and disease. Further examples of advances in both plant and animal breeding could be given almost indefinitely.

Medical Applications

It is well known that the rare phenomenon of mutation can be considerably speeded up by *x*-ray irradiation. Various strains of the mould *Penicillium* were irradiated and a search was made for high-yield mutants. The process was then repeated, with the result that in less than a year a fivefold to sevenfold increase in the yield of penicillin was obtained under standard growth conditions.

Genetics has also something to say about another aspect of antibiotic therapy. The genetic study of bacteria indicates that strains resistant to penicillin, streptomycin, and sulphonamide drugs may originate through mutation. The antibiotic eliminates the susceptible bacteria and gives the resistant mutants a chance to survive and multiply. A thorough understanding of

this mechanism will make it increasingly easier to plan the clinical use of antibiotics in such a way as to minimize the risk of the development of resistant strains.

Although one of the essential ingredients of the evolutionary process is the variability provided by mutation, most mutations are deleterious for organisms which are fairly well adapted to a steady environment. Under ordinary conditions it can be shown that most fully recessive mutant genes that are capable of detection will lie latent for thousands of years before becoming manifest. However, heavy irradiation of the germ-plasm can occur through faulty technique in radiology and is an even greater danger with the use of atomic energy in peace and war. This may result in a very great increase in the natural mutation rate, producing an appreciable deterioration in human heredity. It is a sobering thought to consider that an atomic bomb falling in a highly populated region will probably cause more deaths in succeeding generations by genetic damage than it will at the time of the explosion by blast, heat flash, and irradiation.

A word should be said about the possibility of using experimental animals for the study of comparatively rare inherited defects. For example, canine haemophilia is a sex-linked recessive condition, as is human haemophilia, and it is also very similar clinically. Again, haemolytic disease of the newborn, due to incompatibility between the bloods of mother and offspring, also occurs in certain animals, notably mules, horses, dogs, and rabbits. But so far the analogy with man is not complete, injury in animals occurring only after birth through the absorption of maternal antibody from the colostrum. When genetic and clinical conditions are sufficiently close in both man and a suitable experimental animal, it is to be hoped that the far greater numbers of the latter available may simplify research and lead to substantial advances.

Much of the fascinating work in medical genetics has been done on rare abnormalities such as haemophilia or Huntington's chorea. The number of different conditions that have been studied is very large indeed, and it is most unlikely that the average student or clinician will ever personally encounter more than a small proportion of them. However, several characters, particularly serological ones, are by no means uncommon.

There are now eight main blood group systems: ABO, MNS, P, Rhesus, Lutheran, Lewis, Kell, and Duffy, which are apparently inherited independently of each other. Thus, with these and the attribute of sex and the ability or inability to taste phenylthiocarbamide (P.T.C.), we may have as many as 10 of the haploid number of 24 chromosomes of man distinctly marked. In searching for linkage these are the characters most worthy of examination. It is difficult to over-emphasize the importance of genetics in serology, and the extreme relevance of this field to everyday problems confronting practitioner and specialist alike. Common applications are to problems of relationship and disputed paternity.

Medico-legal Work

One of the best-known elementary cases is that of Mrs. W, who found on returning home from hospital that her baby had on its back a piece of adhesive tape bearing the name "B." The baby at the "B" house was labelled "W." Had the babies been interchanged or had someone been careless with the labels? The bloods of the six persons involved were as follows:

	Group	Genotype		Group	Genotype
Mr. B.	AB	G^AG^B	Mr. W.	O	gg
Mrs. B.	O	gg	Mrs. W.	O	gg
Baby "W"	O	gg	Baby "B"	A	G^AG^A or G^Ag

In this table G denotes a dominant gene and g a recessive one. When the pair are identical, they are said to be homozygous, when different (as G^Ag) they are called heterozygous. A recessive gene is one which cannot make its influence felt in the individual when present with a dominant, but which after segregation (separation from its dominant partner) may have the chance to manifest itself in the offspring.

There had clearly been an accidental interchange of babies before the mothers went home. This is doubly proved, for Mr. B could not have had an O child, nor could Mr. and Mrs. W, both of group O, have produced an A baby. In this extreme case no recourse to any other group is necessary. The same kind of principle applies to disputed paternity. Although a decisive result is by no means always the case, the use of as many blood groups as possible makes such a result very much more likely. In this country extensive use of the available knowledge about the inheritance of blood groups has not yet been made, although considerable steps have been taken towards this in the United States. Only recently a court refused to accept quite definite evidence which clearly disproved paternity. It seems likely that about 50% of the men against whom affiliation orders have been made have been wrongfully accused. It can be shown that by recourse to the full series of eight blood groups now available about 62% of those wrongly accused could be exonerated. As about 7,200 affiliation orders are made every year and 2,000 men go to prison for non-payment, it seems that preventable injustice is occurring on an appreciable scale

It is usual for most textbooks to say that paternity tests can only be negative—that they can only on occasion disprove paternity, and can never provide positive evidence in its favour. This was the case when knowledge of blood groups was in its infancy, but is no longer true with the existence of rare antigens or combinations of antigens For example, suppose a woman of rhesus constitution cde/cde has a baby with the extremely rare genotype C^wde/cde, while the putative father is also C^wde/cde. The conclusion would be irresistible—though perhaps not in a court of law. In a similar manner a number of rare abnormalities might occasionally give positive evidence of paternity. In 1921 there was reported in Norway a paternity case in which a normal mother had given birth to a brachyphalangic child. Brachyphalangy is inherited as a rare simple dominant, and the court accepted the fact that the putative father was brachyphalangic as evidence of his paternity.

Now evidence concerning blood groups may be required by either party in divorce or kindred proceedings, and a doctor called as an expert witness may find himself exposed to the following criticism. He may be asked, "Is it not true that genetic factors are subject to sudden and unpredictable changes, called mutations, and is not evidence depending upon their inheritance therefore suspect? Can it be asserted that the genetic factor in question has not in fact changed in this way?" It is essential to be prepared for such opposition, and it is worth while giving a correct reply here in some detail. It would run something like this.

The hereditary mechanism of all higher organisms is of the particulate or Mendelian type, which depends for its

operation upon the extreme permanence of the hereditary factors themselves. Were these to change or mutate in as many as one individual in even a few thousand, the system would break down. A study of such mutations throughout the widest selection of living organisms demonstrates that a mutation rate of as high as 1 in 50,000 individuals is extremely rare, and this frequency is hardly ever exceeded. Inheritance in man is of the same type, and these conclusions are strictly applicable to him. However, it is unnecessary to depend on mere analogy for a knowledge of human mutation rate. It has been possible to study this in several instances in which it proved to be, when most frequent, of the same order of rarity as in other animals. The error introduced into blood grouping by mutation is very unlikely to be greater than 1 in 50,000, and is probably less.

Few legal cases can have been decided in the present century in which the possibility of mistake was not considerably greater than this.

The Rhesus Group and Haemolytic Disease

The importance of avoiding serological incompatibility in blood transfusion is well known. With the ABO group one merely has to ensure that the transfused erythrocytes are not agglutinated by the recipient's antibodies. With the rhesus group, on the other hand, the danger is that a rhesus-negative woman may become immunized by transfusion or pregnancy, with the consequent risk of haemolytic disease of the newborn in her offspring. The practical implication that every female requiring transfusion, or who has or who may become pregnant, should be rhesus-tested does not of itself involve genetics. But in the case of a rhesus-negative woman for whom one wishes to assess possible risks the invocation of genetical principles is essential.

It should be noted that the great majority of cases of rhesus incompatibility are in connexion with the antigen D, although the others (C,E ; c,d,e, etc.) may very occasionally stimulate the production of antibody.

When a rhesus-negative woman marries a rhesus-negative man there is no danger, as all the children will be rhesus-negative. (The mating is of type dd × dd.) Even if she marries a rhesus-positive man (dd × Dd or dd × DD) the risks are still small, in spite of the contrary assertions of newspaper articles. Provided the woman has never been transfused or injected with even minute amounts of rhesus-positive blood the chance of the first child suffering from haemolytic disease is almost zero, and the chance for the second child still extremely small. The combined chance of disease in the first four or five children is uncertain, but is perhaps no greater than 5% if the husband's genotype is unknown. With homozygous (DD) husbands this chance would be about 9%, and with heterozygous husbands (Dd) it would be only 2%.

If, however, the woman has already given birth to an affected infant, further rhesus-positive children are very likely to be diseased and only rhesus-negative children will be healthy. If the father is heterozygous, then there is an even chance of a healthy child at each subsequent pregnancy. On the other hand, if the father is homozygous, all future children are likely to be affected ; but, even so, there is still some chance that a child diseased at birth will respond to treatment and eventually be healthy.

Collecting Data and Preparing Pedigrees

It is a peculiar characteristic of genetic data that they are often collected only incidentally during the course of work primarily concerned with some other department of medicine. More pedigrees of rare inherited abnormalities have been placed on record by physicians in the course of their ordinary work than have been amassed by research workers making special efforts to obtain such material. A lot of information of a genetical nature normally arises in clinical, pathological, and socio-medical investigations.

Carefully and properly collected genetic data have a certain permanence about them which is not so characteristic of other branches of medicine where new methods of observation and experiment frequently make older work obsolete. Although the systematic and detailed construction of pedigrees is of fairly recent origin, it is likely that they will be of immense value for centuries to come.

Desiderata for Good Family Record

Let us consider some of the special points to be observed in keeping a good family record. Clinical histories of individuals are usually perfectly adequate for their immediate purpose. But when their information on hereditary factors is later extracted and collated it is only too common to find that vital links in the chain of inference are missing because their relevance was not apparent at the beginning.

While there is already in existence a vast but relatively untapped mine of information in the records of general practitioners which could be made to yield valuable results if systematically investigated, it is also certain that a great deal of promising data would in fact be found to be of little use because of the failure to observe one or two elementary precautions in the original compilation.

Obviously, in reporting an abnormality or disease of any kind it is essential to have a full clinical description, including such items as the age and sex of the patient, age at onset of the disease, variations in severity and frequency of attack, and so on. Some indication of the patient's social background should also be given ; his social and economic status, the type of work he does, etc. It is also important to have this sort of information about near relatives, especially parents, brothers and sisters (sibs), and children. It is these near relatives that are the most important—data should always be obtained about more distant relatives if available, but this is not quite so essential.

The usefulness of a family history is naturally dependent on the firm establishment of the clinical diagnosis, and in the case of the patient through whom the pedigree first comes to be noticed (called the "propositus") this will normally have been obtained at first hand. It often happens that the doctor will be able to see the close relatives himself and record his own direct impressions—this is much the most satisfactory state of affairs. But sometimes it is possible to secure quite good second-hand information from the patient himself or other relatives—for example, about members of the family whom the doctor is unable to see himself. In an extensive study it might be useful to classify the individuals in some such grouping as : "certainly affected," "almost certainly affected," "possibly affected," "almost certainly unaffected," "certainly unaffected," "unknown," etc. But in any event there should always be notes on each individual giving the exact evidence itself. Should it prove impossible to obtain information about any particular relative of the generations and degrees of

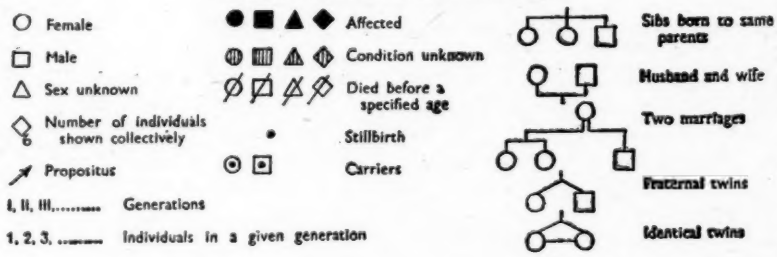
kinship studied, the reason for the failure should always be stated.

When collecting together data on several related persons in this manner one should clearly indicate who is the propositus—the individual who first brings the family to notice, or primary case in the genetic sense. Failure to do this may seriously affect the interpretation of genetical data, particularly in the sib method of analysis, which is discussed below.

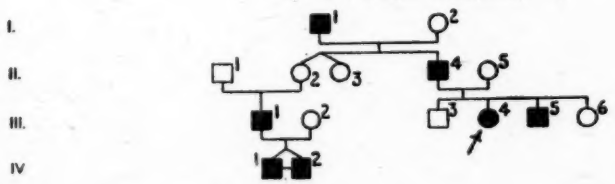
Another vital piece of information which must not escape the record is the existence of a consanguineous marriage. Positive or negative information on this point is equally valuable. In many cases it is not sufficient merely to state that husband and wife are related, that they are cousins, for example : one wants to know in greater detail, for instance, whether the mother of the husband and the father of the wife are brother and sister. Cases of twins should also be carefully noted, together with any positive or negative information on whether they are identical or not.

It is useful to be able to lay out an interesting pedigree so that the salient characteristics may be seen at a glance. There are various ways of doing this, and some of the main symbols in common use are shown in the accompanying Table. In complicated pedigrees, especially

Pedigree Symbols in Common Use



Part of a pedigree of polydactyly



those involving the inheritance of two or more characters, special symbols may have to be employed. For further suggestions, reference may be made to Ruggles Gates's *Human Genetics,* vol. 1, p. 4.

Ascertainment

The modern methods employed for interpreting genetical data normally involve some kind of statistical analysis. The reliability of these analyses depends on a knowledge of the way in which the material was collected—what is usually called the " method of ascertainment." Uncertainty about ascertainment will affect the validity of the results. Sometimes it is possible to make some allowance for ignorance of the method of ascertainment, but naturally there is an inevitable loss of certainty.

One might in theory collect all the cases of a particular condition in a given neighbourhood or district. Such a procedure would readily lend itself to simple interpretation, but would in general be exceedingly troublesome to carry out. It is usually possible to gain experience of only a certain proportion of all the available cases. If one collects consecutive cases of a condition, then the correct proportion of affected individuals among sibs (which is the most informative figure) is obtained by calculating the proportion among the remainder of each sibship, ignoring the propositus. If two members of a single sibship present themselves independently then that sibship must be counted twice over. For example, if a sibship consists of four normal and three affected individuals, of whom two of the affected persons appear for examination independently while the third is found only as a result of a family inquiry, then that sibship is counted twice, each time as four normal and two affected.

Two points stand out. First, that the right method of analysing the data must be employed. Second, that useful information can be obtained from a study of sibs only. It follows, therefore, that there is no need to be discouraged because of the difficulty of seeing or gaining information about distant relatives. It is worth making a special effort to obtain detailed information about parents, sibs, and children, but less than this can be of use. The same thing is true of data which are collected in the hopes of finding a genetic linkage. There is not space to go into these methods now ; suffice it to say that the general principle is to test the propositus, together with his sibs and parents if available, for the possession of suitable characters other than the one used for selection—say, the individuals' blood groups or their ability to taste P.T.C., etc.

Eugenic Prognosis

A few comments on the practical value of a knowledge of linkage may be mentioned here. Unfortunately such knowledge is still somewhat rudimentary in man, but with the increasing number of marked chromosomes, especially those due to the discovery of new blood groups, quite extensive data on human linkage are potentially within our grasp.

Many serious human defects are heterozygous conditions, and some of them do not become apparent until after the normal age of marriage, as in Huntington's chorea. Children whose father or mother has developed that disease may wish to marry. Though they are apparently healthy it is probably undesirable for them to do so if between the ages of 30 and 40 they are destined to develop a progressive mental deterioration leading to insanity, which they will moreover transmit to half their offspring. At present such individuals can only be told that the chances are exactly equal: they may be afflicted in this way, or they and their descendants may remain perfectly normal. If, however, we knew of a number of genes controlling ordinary characters, such as eye colour, blood groups, P.T.C. tasting, attached ear lobes, etc., which were linked with Huntington's chorea, then the situation would be much clearer. We could say perhaps whether the chances of developing the disease or not were very remote or very considerable, and this might have far-reaching effects on the decisions of the patients concerned. Similarly, in families with sex-linked disease it would be invaluable to know whether a woman was carrying a recessive gene or not.

It is commonly said that in families harbouring a serious recessive defect no one should marry a near relative, that marriages between cousins should be prohibited. While the ethics of this sort of problem are by no means easy, an assessment of the probabilities involved may well provide useful guidance. If there is any doubt about how to do this the advice of a competent geneticist should be sought.

Suppose, for example, a man has had a brother who died of juvenile amaurotic idiocy, which is due to an autosomal recessive gene (one not on a sex chromosome), and that he wishes to marry his first cousin. If there are no other cases of the disease in the family then the chance that both the man and his cousin are heterozygous is 1 in 6. The chance of a single child from such a marriage being an idiot is therefore 1 in 24. The couple may be prepared to accept this risk, but it is desirable that they should know what it is. The chances of having an affected child are greater if they have more than one; and the probability of producing a heterozygous child, which though itself unaffected is capable of transmitting the gene, is considerably higher. In any case a consanguineous marriage always increases the likelihood of bringing to light an undesirable recessive gene.

To take another case, consider a woman who has a brother who dies of true haemophilia, which is a sex-linked recessive. What are the prospects both for her and for another brother who is unaffected? The unaffected brother cannot carry the gene at all and his descendants will all be free of it. The woman, on the other hand, has a 50% chance of carrying a gene for haemophilia. If this is so she will transmit the disease to half her sons, and half her daughters will be carriers.

It is interesting to note that the linkage of the loci (gene positions) for haemophilia and colour-blindness has already been used for eugenic prognosis in families exhibiting both these defects. In a certain Dutch family it was possible to say that the colour-blind daughter had a 90% chance of carrying a haemophilia gene, where her normal sisters had only a 10% chance. Probabilities like these are clearly of much greater value than the more usual 50% chances, as in the previous example. No doubt when the human germ-plasm is as well charted as that of *Drosophila melanogaster*, this kind of argument will be used as a matter of course in the prevention of congenital abnormalities.

Aids to Diagnosis and Preventive Medicine

The discovery of a hereditary factor in the aetiology of a disease by no means implies that further medical progress is impossible. Heredity and environment interact and determine jointly the condition of a person. Thus an ailment which depends to some extent on heredity may be ameliorated, cured, or even entirely avoided by suitable treatment or alteration in living conditions.

There are occasions when a knowledge of hereditary background can be used as an aid to diagnosis. For instance, a man was admitted to hospital having lost considerable blood by haematemesis. The diagnosis lay between gastric ulcer, cirrhosis of the liver with oesophageal varices, and Banti's disease. One physician, however, happened to know that the man's father had suffered from a mild telangiectasis and had had the typical spider's-webbing of the nasal mucous membrane. Now, multiple telangiectasis, or Osler's disease, is due to a dominant gene, and it was suggested that perhaps the patient in question also had the disease—with a telangiectatic spot in the gastric mucosa. The surgeon explored, found a large telangiectatic focus and excised it. Symptoms disappeared and there was no recurrent haemorrhage. If a dominant gene always has full expression then it is true that no treatment will prevent the bearers from exhibiting the abnormality; nevertheless some form of treatment may be available, as in the surgical treatment of polydactyly.

In other circumstances genetic knowledge may help to detect hereditary diseases in their preclinical stages, when full advantage can be taken of preventive measures to forestall the appearance of manifest disease. Thus familial acholuric jaundice is a dominant condition for which splenectomy is a common prophylactic measure. Moreover, it can always be recognized preclinically by certain laboratory tests. Although it would not be feasible to examine whole populations of individuals, special examination can be made of the relatives of known sufferers. When laboratory tests show an individual to be carrying the gene, the spleen can be removed, if indicated, and clinical disease probably averted

Conclusion

It can be said with full justification that a deeper understanding of the genetical basis of a disease will often help to fill in the gaps in a purely clinical approach and may make material contributions to both treatment and prevention. The field of application of genetics to medicine is a wide one. There is the study of mutation, which has been applied to the advancement of antibiotic therapy and which has also emphasized some of the dangers attached to *x*-ray irradiation and the use of atomic energy. There is the study of the inheritance of blood groups and other common characters as an aid to identifying individuals, particularly in paternity cases. The rhesus blood groups are of special interest, and genetical theory is particularly valuable in estimating the risks run by rhesus-negative mothers. The possibility of gaining new insight into various diseases in man by an investigation of closely similar diseases in experimental animals is also worthy of consideration. Genetics can be used as an aid to diagnosis, and for suggesting certain procedures in preventive medicine. Another important application is to eugenic prognosis, when one wishes to assess some of the dangers attached to parenthood where there is the possibility of heritable disease.

There can be no doubt that medical genetics has a very real contribution to make to the cause of suffering humanity, and that its power and scope will greatly increase and spread in the years ahead.

In his annual report for 1949 Dr. Hugh Paul, medical officer of health for Smethwick, writes: "It is unfortunate that many of these [investigational] facilities are only available to the practitioner through a senior member of the hospital staff; for example, the general practitioner may find great difficulty in getting the result of an examination of the patient's blood, a bacteriological examination, or a radiograph, except by making an appointment with the appropriate consultant in the hospital. This may be excellent hospital etiquette, but it is deplorable medical practice, and the technical facilities of the hospital, particularly those associated with bacteriology and biochemistry, should be immediately available to the general practitioner without the intervention of a third person except when the general practitioner asks for an opinion as well as for a test."

AN INVESTIGATION OF THE PROPERTIES OF ISOPROPYL CHLORIDE

BY

JOHN E. ELAM, B.A., M.R.C.S., L.R.C.P.
Anaesthetist, Barnet General Hospital

AND

M. L. NEWHOUSE, M.B., M.R.C.P.
Assistant Physician, Barnet General Hospital

MacDonald (1950) has described the anaesthetic properties of isopropyl chloride ("proponesin"). This agent has now been used by one of us (J. E.) in approximately 50 cases and has been found to have all the properties that anaesthetists have long been waiting for. The induction was pleasant for both anaesthetist and patient, and with only a gas–oxygen–isopropyl-chloride sequence the most resistant patient could be smoothly led to the deepest plane of anaesthesia without cough or struggle. This anaesthetic seemed to be equally successful for both major and minor surgery; good muscular relaxation was provided and recovery was both rapid and pleasant. It was noticed that cardiac irregularities occurred, but, as these are not uncommon with sodium thiopentone, "trilene," and cyclopropane, they were disregarded. In order to avoid these irregularities, several patients were induced with gas, oxygen, and ether, and then a change was made to isopropyl chloride, but cardiac irregularities were still noticed in each case, though the patient suffered no ill effects. Unfortunately, a case of complete circulatory failure occurred.

The patient, a man aged 48, was a case of moderately severe thyrotoxicosis. Before operation he had had five weeks' rest in bed and three weeks' premedication with Lugol's iodine. Electrocardiograms taken three weeks before operation showed sinus tachycardia but no evidence of myocardial damage. Under treatment he had gained 5 lb. (2.3 kg.) in weight and his pulse rate fell from a range of 120–110 to 90–80. For the induction of anaesthesia the nitrous-oxide–oxygen–isopropyl-chloride sequence was used. The induction was very smooth and quiet. The patient's colour was good throughout and at no time was there any indication that some difficulty might arise. An endotracheal tube was passed very easily and the patient taken into the theatre. Just 10 minutes after the induction was started, as the patient was being lifted on to the table, he gave a slight cough and his colour changed to ashen grey; he was found to have complete cardiac arrest. Controlled respiration with oxygen was continued throughout the attempts to restore him. Cardiac massage with intracardiac injections of adrenaline and procaine were given, and after 45 minutes the heart was restarted, but he died a few hours later (Roualle, 1950).

After this experience it was decided that electrocardiographic records should be obtained during the administration of that anaesthetic. For this, two healthy young men were chosen. Only nitrous oxide, oxygen, and isopropyl chloride were used.

Case 1.—The patient was a man aged 31, and the operation was for the removal of a semilunar cartilage. The preliminary record was normal (Fig. 1); thereafter records were taken at approximately five-minute intervals, and more often when the pulse was irregular (Fig. 2). After five minutes Lead II showed a regular rhythm, but with suppression of P waves and depression of the S–T interval; at 11 minutes a curious rhythm had developed with dissociation of the auricular and ventricular elements. The P waves were absent for several beats and then appeared with no constant relation to the QRS complex; there was a high degree of A–V block and possible nodal rhythm. At 17 minutes the rate was 200 a minute, and there were multifocal ventricular extrasystoles; at 19 minutes the rate was 100 and irregular, with absent P waves, suggesting auricular fibrillation. A continuous record was then taken, which showed variations between multiple ventricular extrasystoles to a coarse auricular fibrillation. Five minutes later he reverted to a normal rhythm, which continued to the end of the operation.

Case 2.—This patient was a man aged 25, and the operation was for the repair of the flexor tendons of the right hand. A normal record (Fig. 3) was obtained before the administration of the anaesthetic. Under the anaesthetic he showed less severe changes than the first patient, but (Fig. 4) at five minutes the rate was 120 a minute, with multiple extrasystoles and depression of S–T intervals in some complexes. Extrasystoles were still appearing in the record taken at seven minutes. At 10 minutes the pulse rate had dropped to 60 and was still slightly irregular, but there were normal contractions. At 25 minutes and at the end of the operation, after 45 minutes' anaesthesia, the rhythm was normal.

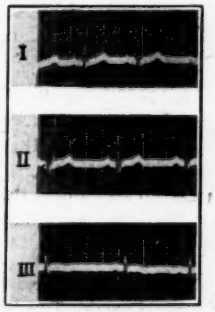


FIG. 3.—Case 2. Pre-anaesthetic record, normal tracing.

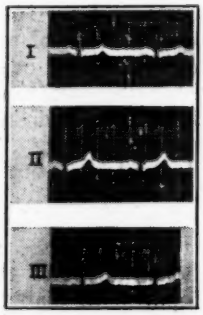


FIG. 1.—Case 1. Pre-anaesthetic record, normal tracing.

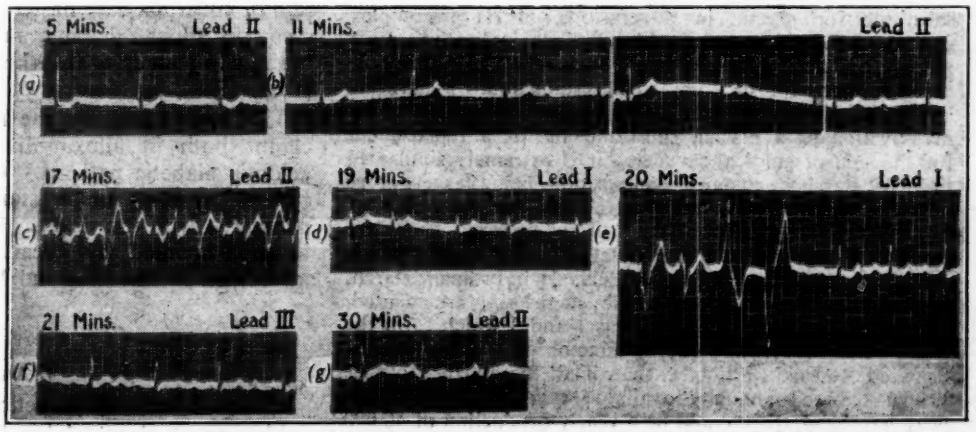


FIG. 2.—Case 1. Consecutive records while under anaesthesia. (*a*) Depression of S–T interval and absent P waves. (*b*) Dissociation of P waves and QRS complexes. Possible nodal rhythm. (*c*) Multifocal ventricular extrasystoles; rate 200. (*d*) Auricular fibrillation; rate 100. (*e*) Ventricular systoles, with two beats showing increasing P–R interval. (*f*) Coarse auricular fibrillation. (*g*) Reversion to normal rhythm.

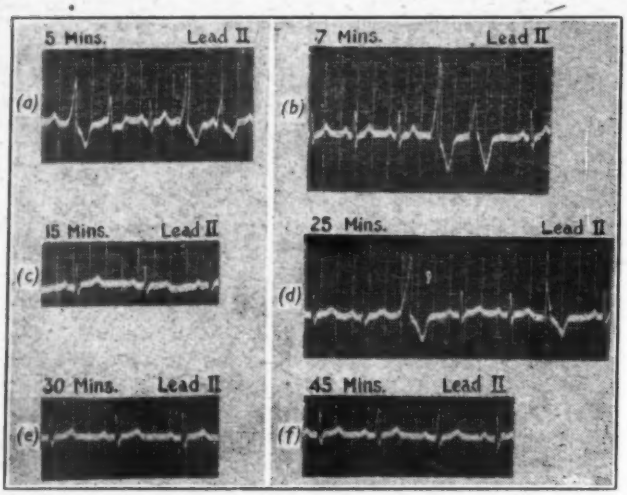


FIG. 4.—Case 2. Consecutive records while under anaesthesia. (*a*) Ventricular extrasystoles with depression of S–T intervals in some complexes; rate 120. (*b*) Ventricular extrasystoles with suggestion of coupling. (*c*) Normal rhythm. (*d*) Patient "light." Recurrence of extrasystoles, without depression of S–T intervals. (*e*) Normal rhythm. (*f*) Normal rhythm.

There is felt to be evidence that isopropyl chloride exerts a direct toxic action on the myocardium, and extreme caution should be shown in its use. Flagg (1939) reminds us that too many people have tried too often to avoid the use of ether, and it is open to question whether, perchance, in casting out the admittedly unpleasant devils of ether and chloroform we are not making way for seven other far worse devils.

Summary

The pleasant qualities of isopropyl chloride are emphasized.

The high incidence of cardiac irregularities are noted and a death under this anaesthetic is recorded.

Electrocardiographic studies showing severe though temporary changes during the administration of the anaesthetic have been made.

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Dextran has already received some notice as a possible substitute for plasma in transfusions. It is a complex gummy carbohydrate synthesized by certain bacteria from glucose, and according to a recent press report plans are now going forward for its commercial production. Meanwhile laminarin, a polysaccharide from seaweed, is also under investigation as a plasma substitute and as a replacement for the talc of surgical powder. It is extremely similar to ordinary starch in chemical composition, being largely composed of glucose units linked in chains, but the points of glycosidic linkage differ in the two substances. There are five possible points on the glucose molecular ring at which other glucose molecules can be attached: in ordinary starch, positions 1 and 4 on opposite sides of the ring are so used, whereas in laminarin it is positions 1 and 3 which form the links. Dr. F. M. Woodward, director of the Institute of Seaweed Research, estimates that, if all the 200,000 tons of brown seaweed annually available round Scottish coasts were harvested, there could be a yearly production of 40,000 tons of laminarin. At the same time about 30,000 tons of mannitol would be available, and roughly the same amount of alginic acid, used for making synthetic fibres for the textile industry.

RAISED BLOOD PYRUVIC ACID LEVEL IN DIABETIC ACIDOSIS

THE VALUE OF COCARBOXYLASE IN TREATMENT

BY

I. C. GILLILAND, M.D., M.R.C.P.
Medical Tutor, Postgraduate Medical School of London

AND

M. MENCER MARTIN, M.B., M.R.C.P.
Formerly Lund Research Fellow, Postgraduate Medical School of London; Diabetic Department, King's College Hospital

Ketosis is a familiar early complication of severe and uncontrolled diabetes mellitus : its causation is understandable, but the steps by which, unchecked, it progresses to coma and shock are not all understood. Usually the standard treatment with insulin and fluids will rapidly resuscitate a patient from diabetic coma : but recovery may be slow or even fail to take place if treatment has been delayed. Prolonged severe diabetic ketosis is evidently complicated by functional disturbances other than those reversible by insulin. One such complication which has recently been clarified is the depletion of potassium and phosphates in the tissues (Guest, 1942 ; Martin and Wertman, 1947). That disordered carbohydrate oxidation may be another is suggested by the recent finding of a raised pyruvic acid level in the blood in diabetic acidosis (Markees and Meyer, 1948 ; Markees, 1949).

Pyruvic acid is believed to occupy a central position in the metabolism of carbohydrates ; it is an intermediate product in the body's oxidation of these and other metabolites. The first step in the oxidation of pyruvic acid is effected by the pyruvic oxidase enzyme system, which is active only in the presence of certain co-enzymes, including cocarboxylase (Banga, Ochoa, and Peters, 1939). Accumulation of pyruvic acid in the blood suggests a failure of this enzyme system, as is the case, for example, in aneurin deficiency, which results in lack of cocarboxylase (vitamin B_1 diphosphate).

Markees and Meyer (1949) and Boulin *et al.* (1949) have suggested that the administration of cocarboxylase may be a useful supplement to the standard treatment of diabetic coma. They have claimed that the addition of cocarboxylase hastens the recovery from diabetic coma in alloxan-diabetic rabbits (Markees, 1949) and in some human cases (Markees and Meyer, 1949 ; Boulin *et al.*, 1949), but control data are not given.

This report presents the results of testing the above claims both in alloxan-diabetic rabbits and in four acidotic diabetic patients. Control observations of the rate of recovery under standard treatment have been compared with the results obtained when this treatment is supplemented by cocarboxylase and riboflavin as advocated by Markees and Meyer (1949).

Method

I. Animals

Plan.—After the induction of alloxan diabetes the rabbits were observed for the development of diabetic acidosis. Eight to ten days after the administration of alloxan a number of the rabbits which had become diabetic gradually passed into a phase of progressive

diabetic acidosis with ketosis and a raised blood pyruvic acid level. The first signs were usually the presence of acetone in the urine ($FeCl_3$ test) and in the breath, and a slowing of respiration. When, usually 12 to 24 hours later, the animals were clearly noticed to be ataxic or unable to stand, they were classed as being in "diabetic precoma." Pairs of rabbits were selected that had reached approximately the same stage of precoma at about the same time after alloxan administration; the treatment to be tested was then instituted. Observations were carried out over an eight-hour period. If one of a pair died during that time the pair were rejected from further observation. Each pair received identical general treatment: 10 to 50 units of soluble insulin intravenously, repeated subcutaneously after two hours, and 100 ml. of normal saline intravenously two-hourly for the first four hours. In addition, one of each pair was given two-hourly 25 mg. of cocarboxylase intravenously and 10 mg. of riboflavin subcutaneously. Blood samples were withdrawn by cardiac puncture every two hours after the onset of treatment. The collection of blood from an ear vein was found to be too slow and unreliable for frequent sampling (Friedemann and Haugen, 1943).

Preparation of Diabetic Animals.—Rabbits weighing between 2,000 and 2,500 g. were used in all experiments. They were kept in metabolic cages and were fed on a constant diet with water *ad lib.* Diabetes was induced (Duffy, 1945) by giving the rabbits 200 mg. of alloxan per kg. body weight in a 4% solution intravenously, the solution being almost neutralized with NaOH immediately before use. A 5% glucose drink was offered for the subsequent 24 hours and given by stomach tube when hypoglycaemic symptoms were noted.

II. Patients

Four patients in diabetic acidosis were tested—two were mildly acidotic and two moderately to severely acidotic, though not actually comatose. All four patients were dehydrated; the two more acidotic patients were vomiting and one was drowsy. The breath of each of the patients contained acetone, and the urine was loaded with sugar and acetone ($FeCl_3$ test).

The two severely acidotic and the two mildly acidotic patients were paired and each pair was given identical treatment. The severely acidotic patients received subcutaneously 50 units of soluble insulin four-hourly and the less acidotic patients 20 units of soluble insulin four-hourly with saline in addition as required. One of each pair was also given 200 mg. of cocarboxylase (vitamin B_1 diphosphate) intravenously and 10 mg. of riboflavin subcutaneously every two hours.

The blood samples from the two mildly acidotic patients were estimated only for pyruvic acid.

III. Estimations

The blood samples from both rabbits and patients were analysed for the alkali reserve by the method of Van Slyke and Cullen (1917), for blood sugar by the Harding modification of the Schaffer–Hartmann method (King, 1947), and for pyruvic acid by the method of Friedemann and Haugen (1943).

Results

Rabbits.—No difference was observed in the rate of recovery from acidosis between rabbits treated with cocarboxylase and riboflavin and those not so treated. All the rabbits that survived lost their ataxia and became more lively, and fed themselves by the conclusion of the eighth hour of the experiment. There was no observable difference between the pairs in these clinical respects. Data of blood pyruvic acid, alkali reserve, and blood sugar are shown in Fig. 1.

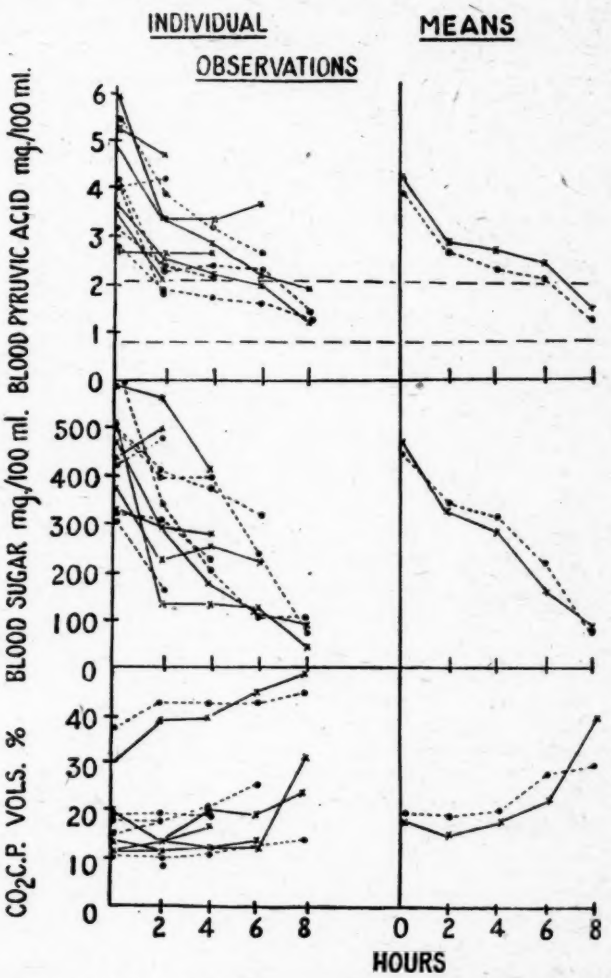

FIG. 1.—Fall in level of pyruvic acid and blood sugar, and rise in alkali reserve in individual rabbits, as well as the means of observations on cocarboxylase-treated and on control animals. ×——× = control animals; ●——● = cocarboxylase-treated animals.

In non-acidotic diabetic rabbits the mean blood pyruvic acid level was found to be 1.32 mg. per 100 ml., with a range of 0.81–2.1 mg. (12 obs.), which compares well with a mean of 1 mg. and a range of 0.3–2.2 mg. reported by Markees and Meyer (1949).

Patients.—The observations on the rate of recovery in the four patients are shown in Fig. 2. No difference, either clinically or in the measurements, was observed in those given cocarboxylase and riboflavin. This might conceivably have been because our patients were not comatose.

Discussion

Although in controlled diabetes mellitus both the blood pyruvic acid level (Bueding *et al.*, 1942) and the cocarboxylase level (Goodhart and Sinclair, 1940) are normal, our findings corroborate those of Markees and Meyer (1948, 1949), that the blood pyruvic acid level is raised in diabetic acidosis both in alloxan-diabetic rabbits and in diabetic patients. But we have been unable to confirm their claim that the giving of

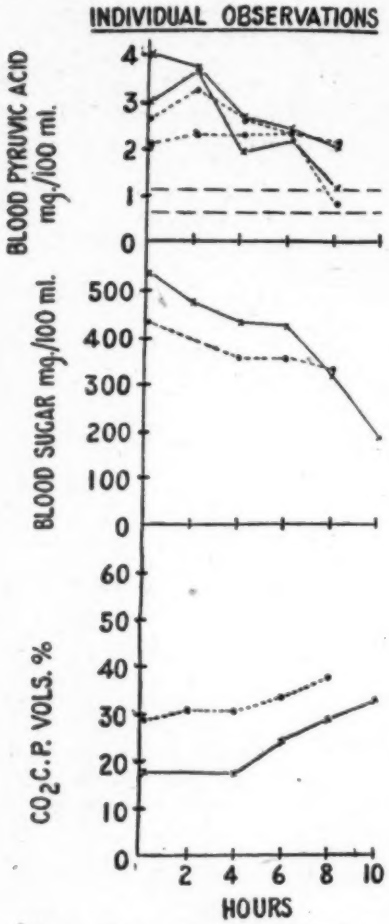

FIG. 2.—Fall in pyruvic acid levels in four patients and fall in blood sugar and rise in alkali reserve in two patients in which these were followed. ×——× = control patients; ●——● = cocarboxylase-treated patients.

cocarboxylase and riboflavin in the treatment of diabetic acidosis hastens the rate of clinical recovery and the return to normal level of the blood pyruvic acid and alkali reserve. We found the rate of improvement to be similar to that seen in paired controls given identical treatment with fluids and insulin without the addition of cocarboxylase and riboflavin; neither in the 12 alloxan - diabetic rabbits nor in the 4 acidotic diabetic patients was there any difference in the rate of clinical recovery, the fall in blood pyruvic acid and blood sugar, or the rise in alkali reserve.

The test of such a treatment in human patients is, of course, more difficult, since comparisons are valid only when comparable cases are contrasted; this was more easily achieved with the rabbits than with the patients. Thus, while we have confirmed the evidence of defective pyruvic acid oxidation in diabetic acidosis, we found no evidence that this was due to a deficiency of cocarboxylase. Normal pyruvic acid metabolism requires not only cocarboxylase and other co-enzymes but also the integrity of the enzyme protein; it may be that in diabetic acidosis the protein component of the enzyme system is inhibited or partially inactivated as it is in sodium arsenite poisoning (Peters, Sinclair, and Thompson, 1946).

Summary

The blood pyruvic acid level was found raised during diabetic acidosis in 12 alloxan-diabetic rabbits and 4 acidotic diabetic patients.

Supplementing a standard treatment for diabetic acidosis by cocarboxylase (vitamin B_1 diphosphate) and riboflavin did not appear to accelerate further the recovery from diabetic acidosis, as measured by the rate of the fall of the raised blood pyruvic acid level, the fall of the blood sugar, the rise of the alkali reserve, or the observed clinical improvement.

We wish to express our thanks to Dr. Russell Fraser for his help and advice, to Dr. F. Wrigley for suggestions, and to Miss Shirley Looker for her assistance with the biochemical estimations. We are also indebted to the Diabetic Association for the grant of a Lund Research Fellowship to one of us. The cocarboxylase used in the investigation was kindly supplied by Roche Products Ltd.

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ORAL PENICILLIN IN CHILDREN

BY

S. A. DOXIADIS, M.D.
Senior Lecturer in Child Health, University of Sheffield

JOHN L. EMERY, M.D., D.C.H.
Clinical Pathologist; Lecturer in Pathology, University of Sheffield

AND

SHEILA M. STEWART, B.Sc.
Bacteriologist; Demonstrator in Bacteriology, University of Sheffield

(From the Children's Hospital, Sheffield)

It has been shown that satisfactory bacteriostatic blood levels are attained in infants by the oral administration of penicillin (Henderson and McAdam, 1946; Buchanan, 1946; Husson, 1947; Moseley, 1948). The oral use of penicillin in infants has therefore been widely adopted. In older children and adults the conditions for absorption of penicillin from the gastro-intestinal tract are not equally favourable, probably because of the higher gastric acidity.

Charney *et al.* (1945) suggested the use of an antacid, and it was shown (György *et al.*, 1945; Burke *et al.*, 1945; Cohlan *et al.*, 1948; Hildick-Smith *et al.*, 1950) that the combination of penicillin with an antacid is effective in maintaining a bacteriostatic blood level. Hoffman *et al.* (1948) were unable to confirm this. They obtained satisfactory results by giving penicillin three-hourly in a dose of 1,400 units per lb. (450 g.) of body weight. All authors agree that penicillin should be given on an empty stomach to ensure maximal absorption.

Owing to the conflicting reports on the oral dose required and on the value of antacids it was felt that a study of these points was necessary before penicillin could be used by mouth for therapeutic purposes.

Methods

Seventy-six children aged 2–15 years were given penicillin two or more hours after a light breakfast. It was in tablet form, each containing 200,000 units. The dose given varied from 1,000 to 7,000 units per lb. of body weight. Sodium citrate, 3 g. in 1 oz. (30 ml.) of flavoured water, was used as antacid. It was given immediately before or after the penicillin.

The blood and penicillin serum levels were estimated by the capillary-tube method described by Fleming (1943), using whole blood inoculated with Richard's strain of *Streptococcus pyogenes*. The serum used for assay was from capillary blood collected in Wright's capsules. Blood was taken before the penicillin was given and three hours later. In many children blood was also taken at other intervals. A total of 208 test doses were given, involving 632 penicillin assays.

Results

The Table shows the number of children grouped according to the penicillin dosage and the use of antacid, with the numbers in each group having a serum penicillin level of 0.06 unit per ml. or over at various times after the ingestion of the drug.

Number of Children with Serum Penicillin Level of 0.06 unit/ml. or More at Various Times after Ingestion of the Drug

Dose; Units per lb. of Body Weight	Hours After Ingestion of Penicillin										
	Without Antacid							With Antacid			
	0	½	1	2	3	4	6	0	2	3	4
1,000–1,999..								0 of 18		**9 of 18**	
2,000–2,999..	0 of 20	7 of 7	11 of 12	10 of 12	**9 of 20**	0 of 12	0 of 12	0 of 20	9 of 9	**16 of 20**	0 of 9
3,000–3,999..	0 of 20	3 of 3	5 of 5	3 of 5	**13 of 20**	0 of 5	0 of 2	0 of 20	5 of 5	**19 of 20**	1 of 5
4,000–4,999..	0 of 20		6 of 6	5 of 6	**16 of 20**	0 of 6	0 of 4	0 of 20	6 of 6	**19 of 20**	1 of 6
5,000–5,999..	0 of 20		6 of 6	13 of 13	**18 of 20**	5 of 13	2 of 6	0 of 20	1 of 1	**19 of 20**	1 of 1
6,000–6,999..	**0 of 30**		7 of 7	7 of 7	**29 of 30**	7 of 7	2 of 7				

The level of 0.06 unit per ml. of serum was considered the minimum necessary for satisfactory therapy in conditions usually treated with penicillin. In this, as in previous works, the serum level three hours after the ingestion of the drug was arbitrarily chosen for the evaluation of this method of penicillin administration.

It can be seen that in the groups of children receiving less than 4,000 units per lb. of body weight without antacid, only 22 of 40 (55%) had an adequate bacteriostatic level at three hours. Of the 40 children receiving 4,000 to 5,999 units per lb. of body weight without antacid, 32 (80%) had a bacteriostatic level three hours after the administration of the drug. Finally, in the group without antacid, 29 of the 30 children (97%) who received 6,000 to 6,999 units per lb. of body weight had a level of 0.06 unit per ml. or over at three hours.

When antacid was given together with the penicillin, 9 of 18 children (50%) receiving 1,000 to 1,999 units per lb. of body weight, 35 of 40 children (87%) receiving 2,000 to 3,999 units per lb. of body weight, and 38 of 40 (95%) receiving 4,000 to 5,999 per lb. of body weight had a bacteriostatic level three hours after taking the penicillin tablets.

Fifty-seven children received the same dose of penicillin on two occasions, the first time without antacid and the second time some days later with antacid, all the other conditions of the test being exactly the same. In 42 children the bacteriostatic level at three hours was the same on both occasions—namely, either above or below 0.06 unit per ml. (in 3 below, in 39 above). Fourteen children had a level less than 0.06 unit per ml. at three hours without antacid and a level of 0.06 unit per ml. or more when antacid was given. One child had a level of 0.06 without antacid and a lower level with antacid.

Discussion

Although it has been suggested (Tompsett *et al.*, 1949) that the maintenance of bacteriostatic blood levels of penicillin for only a few hours every day may have therapeutic effects, the aim of most penicillin regimens in clinical practice is still the attainment and maintenance of a bacteriostatic blood level for the whole period of treatment. On this basis the results of the present work are discussed.

Hoffman *et al.* (1948) claimed satisfactory results with unbuffered penicillin with single doses as low as 1,400 units per lb. of body weight, and they recommend this dose at three-hourly intervals; but a study of their data reveals that of 137 children aged 1 to 12 years 41 had a blood level of less than 0.03 unit per ml. three hours after the oral dose. With a slightly higher dosage this level was found in 11 out of 85 children.

Similar results with unbuffered preparations and doses of approximately 1,500 to 3,000 units were obtained by Reisman *et al.* (1947), Cohlan *et al.* (1948), and Hildick-Smith *et al.* (1950).

In this investigation an attempt has been made to estimate the amount which had to be given in order to obtain a serum level of at least 0.06 unit per ml. three hours after penicillin was taken. The results show that even when the dose given by other investigators was doubled (4,000 to 5,999 units per lb. of body weight) 8 of 40 children (20%) had at the end of three hours a serum level of less than 0.06 unit per ml.

It was only when 6,000 to 6,999 units per lb. of body weight were given orally that the desired effect was achieved in all but one of 30 children (97%). It would seem that doses smaller than 6,000 units per lb. without antacid cannot be relied upon to maintain a bacteriostatic serum level of at least 0.06 unit per ml. over a period of three hours.

According to Reisman *et al.* (1947) and Cohlan *et al.* (1948) the addition of antacids did not improve the results when 50,000 to 100,000 units were given to children aged 2–12. Hildick-Smith *et al.* (1950), administering a dose of approximately 3,000 units per lb. of body weight with an antacid, obtained blood levels of 0.08 unit per ml. or over in all 10 children at the end of three hours.

The experience from the present series demonstrates the value of simultaneous administration of an antacid. With a dose of 2,000 to 5,999 units of penicillin per lb. of body weight a blood level of 0.06 unit per ml. or over was obtained in only 54 of 80 test doses (67%) without antacid, compared with 73 of 80 test doses (91%) when antacid was given.

The comparison in individual cases of the results with and without antacid, but on the same dose, further demonstrates this point. Of 57 children thus compared, 14 had a satisfactory level at three hours only when an antacid was given, but in one the effect was the opposite.

It is difficult to estimate the magnitude of the economy in penicillin achieved by the addition of an antacid. The results show that, when no antacid is given, at least 6,000 units of penicillin per lb. of body weight must be administered to ensure a level of 0.06 unit per ml. or over at three hours in more than 90% of the children. When an antacid is given the minimal dose likely to have a similar effect, as it appears from the table, is 3,000 units per lb. of body weight. On this basis, the addition of an antacid would permit a reduction of approximately 50% in the dosage of penicillin.

It is generally advised that if penicillin is given orally the dose should be three to five (Poncher and Unna, 1950; György and Lee, 1947) or four to eight (Herrell, 1949) times larger than the parenteral dose. As the recommended single dose for parenteral use in mild and moderate infections is 500 units per lb. of body weight (or 4,000 units per lb. per 24 hours in eight doses) even an eightfold increase in dose is inadequate for oral use. When no antacid is given nothing short of 12 times the parenteral dose can be trusted to produce satisfactory blood levels in most children. This would mean that a 10-year-old child would need more than 3,000,000 units of penicillin a day, or more than 1,500,000 units if an antacid were given at the same time. Another disadvantage is the three-hourly administration. This method of giving penicillin must therefore be weighed against the parenteral injection of a slowly absorbed preparation such as procaine penicillin in 2% aluminium monostearate, which is effective if given once every 24 or 48 hours (Emery *et al.*, 1949).

Summary

Blood levels of penicillin after oral administration of the drug with and without an antacid have been estimated in 76 children, who were given 208 test doses.

To attain a reliable bacteriostatic level of 0.06 unit per ml. three hours after the ingestion the single dose of penicillin should be not smaller than 6,000 units per lb. of body weight—that is, 48,000 units per lb. of body weight in 24 hours.

Simultaneous administration of an antacid may allow a reduction of this dose by approximately 50%.

We would like to thank Mrs. E. L. Henderson, matron of Ash House Hospital School, and her nursing staff for their co-operation and assistance. The penicillin tablets were kindly supplied by Glaxo Laboratories Ltd., Greenford, Middlesex.

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INSTILLATION OF STREPTOMYCIN INTO THE PERICARDIAL SAC IN TUBERCULOUS PERICARDITIS

BY

R. P. K. COE, M.D., M.R.C.P.

Consulting Physician, West Middlesex Hospital; Late Medical Registrar, St. Helier Hospital, Carshalton

Several cases of tuberculous pericarditis treated with streptomycin by the intramuscular route have been reported in the literature. Reference is made in a report of the Medical Research Council (1950) to one case of tuberculous pericardial effusion in which streptomycin was injected into the pericardial sac, but no details or dosage were given. Tapie *et al.* (1950) also report the case of a man of 44 with chronic pulmonary tuberculosis who developed a pericardial effusion. They gave five injections of streptomycin, totalling 1.15 g., into the pericardial sac without obvious improvement.

The following case is recorded because it shows that streptomycin injected into the pericardial sac, in conjunction with streptomycin by the intramuscular route, constitutes a safe and rational form of treatment in tuberculous pericarditis with effusion, and that it may be beneficial, especially in cases characterized by rapid reaccumulation of fluid after paracentesis.

Case Report

A Post Office electrician aged 20, whose brother was suffering from a pleural effusion, was found to have enlarged hilar glands on mass radiography in September, 1948. He was asked to attend for further radiological examination, but did not do so. On January 8, 1949, he developed substernal pain, malaise, fever, and vomiting, and two weeks later was admitted to hospital.

Examination on admission on January 22 revealed a well-built, pale, ill man, moderately dyspnoeic, and with a temperature of 103.2° F. (39.5° C.). There was pulsus paradoxus and moderate engorgement of the neck veins, the apex beat was just palpable in the fifth left space in the mid-clavicular line, and cardiac dullness extended from 1 in. (2.5 cm.) outside the right sternal border to the mid-axillary line. Pericardial friction was heard over a wide area of the praecordium. There was an area of dullness with bronchial breathing and increased vocal resonance over the left lower chest posteriorly near the midline. The blood pressure was 130/70.

Diagnostic pericardial paracentesis on January 26 yielded dark straw-coloured fluid which grew *Mycobacterium tuberculosis* on guinea-pig inoculation, but direct examination and Lowenstein culture were negative.

On January 30 he developed cardiac tamponade, and 32 oz. (907 ml.) of fluid was aspirated, with immediate relief of symptoms. Signs of tamponade returned, however, in 24 hours, and a further 22 oz. (624 ml.) was aspirated the following day. Within the next two months recurrent tamponade necessitated five further aspirations, and amounts varying from 14 to 32 oz. (397 to 907 ml.) were removed on each occasion.

Intramuscular streptomycin, 0.5 g. twice daily, was started on February 12, and, although this resulted in some lowering of the temperature, the pericardial effusion continued to accumulate at about the same rate as before. It was therefore decided to give streptomycin into the pericardial sac. On March 24 1 g. dissolved in 2 ml. of water was injected into the effusion. The procedure was repeated weekly for another five weeks. No adverse effects occurred and after the third injection there was a considerable drop in fever.

The fluid re-formed very slowly, only 6 oz. (170 ml.) being aspirated on two occasions after beginning treatment (see Chart).

On April 21 he developed a left-sided pleural effusion, which was aspirated several times. Intramuscular streptomycin was stopped on May 2 because of toxic symptoms.

On May 3 there were signs of superior mediastinal obstruction and a large mass of enlarged mediastinal glands was seen on radiological examination. Two weeks later ascites, oedema of the legs, and enlargement of the liver to 3 in. (7.6 cm.) below the costal margin were present. All these manifestations were unaccompanied by any notable recurrence of fever or toxaemia, and they slowly subsided with rest in bed over the course of the next six months.

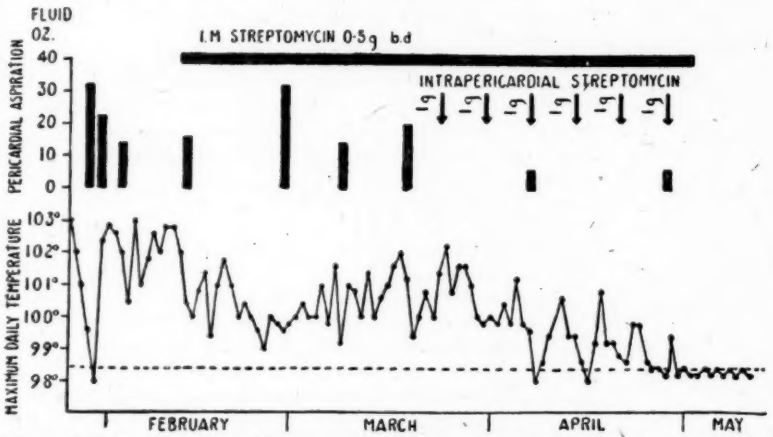

Chart showing maximum daily temperatures, streptomycin therapy, and amounts of pericardial effusion aspirated.

When last seen (May 6, 1950) the patient was extremely well and back at full work, and was able to walk five to six miles (8 to 9.7 km.) at a brisk pace without dyspnoea. Radiologically there was moderate enlargement of the cardiac shadow, with slightly diminished cardiac pulsation, and still some enlargement of the hilar glands on the right side. The blood sedimentation rate was 2 mm. per hour (Westergren, 200 mm.).

I wish to thank Dr. J. B. Harman for permission to publish this case.

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Medical Memoranda

Perforated Peptic Ulcer of Meckel's Diverticulum

A case of Meckel's diverticulum containing gastric mucous membrane and presenting with perforation of an associated peptic ulcer is described. The salient features are discussed, with special reference to the occurrence of peptic ulceration in these cases.

CASE REPORT

A girl aged 7 was admitted to the children's ward of Grantham General Hospital at 1 p.m. on November 14, 1948. She had always been healthy until three days before admission, when she began to complain of diffuse vague abdominal pain. It was noted that the stool soon after the onset of the pain was dark red. On the day before admission two small stools of similar colour were again passed, and on that night the pain became more severe and was definitely colicky in nature, doubling the patient up and preventing sleep. A small amount of yellow fluid was vomited two hours before admission, and the pain then continued as a dull central abdominal ache. There were no urinary symptoms. The bowels had been regular before the onset of the pain.

On examination the temperature was 101° F. (38.3° C.), pulse 140, and respirations 24. The abdomen showed generalized rigidity, maximum over the lower right rectus and right iliac fossa. Tenderness was present over the central part and right side of the abdomen, but there was no hyperaesthesia. Rebound tenderness was also present. There was dullness to percussion in both flanks, and shifting dullness could be demonstrated on moving the patient to one side or the other. Peristaltic sounds were present. There was no tenderness on rectal examination, but a smear of dark-red blood-stained faeces (benzidine +++) was seen on the examining finger. The Hess capillary fragility test was negative, and bleeding and clotting times were normal. A tentative diagnosis of perforation of Meckel's diverticulum was made and operation arranged.

At operation, performed by Mr. F. J. Jauch, the peritoneal cavity was found to contain a large quantity of dark-red blood. Flecks of fibrin were adherent to the lower ileum, and a small Meckel diverticulum measuring 3 by 1 cm. was found arising from the antimesenteric border of the ileum about 18 in. (46 cm.) from the ileo-caecal valve. The diverticulum was adherent to the antimesenteric wall of the intestine for the whole of its length except for the tip, and was covered with flecks of fibrin. At its base there was a small punched-out lenticular perforation measuring ¼ in. (0.6 cm.) in diameter. The ileum distal to the lesion was slightly distended and the coat was bluish in colour. The appendix was normal. No other lesion was detected in the abdomen. The diverticulum was then excised, together with a diamond-shaped cuff of intestinal wall surrounding it. Free dark-red blood was present in the intestinal lumen. The gap was sutured transversely, the excess blood mopped away from the peritoneal cavity, and the abdominal wall closed in layers without drainage.

The patient made a satisfactory recovery from the operation, being allowed up on November 16. A barium meal examination carried out on December 1 showed no abnormality and no delay in the passage of the meal through the small intestine. When last seen, on January 6, 1949, the patient was in good health, eating well, and ready to go back to school.

Pathological Report.—Section shows typical gastric mucosa with numerous well-defined oxyntic cells. At the base of the diverticulum encroaching on both ileal and gastric mucosa there is a subacute type of peptic ulcer which has perforated.

COMMENT

This case is typical in many ways. It falls into the usual age group: 85% of the cases reviewed by Johnston and Renner (1934) were under 16. The perforation was preceded by haemorrhage—this is a common occurrence, 70 to 80% of such perforated ulcers having been associated with haemorrhage. The blood passed per rectum was said to be unmixed with mucus, and this was confirmed by rectal examination. Schullinger and Stout (1934) stress this point in the differential diagnosis from intussusception. The ulcer found at operation was marginal—this is the usual site of ulcer associated with Meckel's diverticulum—and there was an early attempt at walling off the process, a point stressed by Vaughan and Singer (1932).

However, in certain aspects the case differs from the typical picture. Thus, it occurred in a girl: of the cases recorded only 10 to 15% have occurred in females. There was an immediate preceding history of abdominal pain of three days' duration: though this does occasionally occur, it is not common. The acute colicky pain complained of on the night before admission was probably due to haemorrhage into the bowel with distension of the ileum. Gray stressed that there is a sudden change from relative well-being to an acute illness in any acute lesion of Meckel's diverticulum. In my case there was no sudden change and it is difficult to state when the perforation occurred.

My thanks are due to Mr. F. J. Jauch for permission to publish this case and for helpful criticism and advice, to Mr. John Bruce, and to Dr. R. B. T. Baldwin for the pathological report.

DONALD M. HANCOCK, M.B., Ch.B.

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Reversible Papilloedema in Pernicious Anaemia

It is recognized that anaemia may be a factor in the causation of papilloedema, yet reports of its occurrence in Addisonian pernicious anaemia are extremely rare.

CASE REPORT

A ship's boatswain aged 58 was admitted to hospital on February 1, 1949, with severe pernicious anaemia of one month's duration. In July, 1946, he first became ill, but was inadequately treated. His vision had been blurred for three weeks, and three days before admission he noticed swelling of the ankles.

On admission he was gravely ill, grossly anaemic, and slightly jaundiced. There was slight pitting oedema over the sacrum. Enlargement of the liver, spleen, or lymph nodes was not observed. The nutritional state was good and no source of blood loss was discovered. The blood pressure was 120/50. There was no abnormality of the heart, lungs, abdominal organs, or nervous system. The urine contained an excess of urobilinogen but no bile salts or pigments and no albumin.

A blood count on February 2 was: red cells, 1,030,000 per c.mm.; haemoglobin, 4.9 g. per 100 ml.; M.C.H., 37.7 $\gamma\gamma$; white cells, 3,000 per c.mm. Films showed macrocytosis, poikilocytosis, polychromasia, reticulocytes 0.2%, and no malaria parasites.

"Anahaemin" produced a dramatic clinical and haematological response with a reticulocyte "crisis" of 29.2% nine days after the beginning of treatment. On March 19 his blood count was: red cells, 4,880,000 per c.mm.; haemoglobin, 14 g. per 100 ml.—a gain of 3,850,000 red cells and 9.1 g. of haemoglobin in 45 days. On February 27 a thrombocyte count was 533,170 per c.mm., with coagulation time 5 minutes 10 seconds and bleeding time 2 minutes 15 seconds. The sacral oedema had disappeared by the twelfth day of treatment. Serial blood pressures were normal after the first week, and the Hess test for capillary resistance was consistently negative throughout his stay in hospital. He was discharged on April 4 for repatriation to England.

OPHTHALMIC REPORT

On admission both pupils were contracted, ocular movements and intraocular tension were normal, and visual acuity (right 6/24, left 6/36) was not improved by lenses. Bilateral papilloedema was more marked on the right than on the left; venous engorgement and multiple retinal haemorrhages were observed. The arteries seemed to be normal. In both fundi, haemorrhages overlapped the disk margins and, more peripherally, multiple flame-shaped haemorrhages in the nerve-fibre layer were scattered in the nasal and temporal areas. A deep oval-shaped haemorrhage with clearly defined edge was seen one disk diameter supertemporally from the right optic disk. An inverted-comma-shaped haemorrhage projected anteriorly from the lower margin of the left macula, and one disk diameter below this a circular haemorrhage was seen in the deeper retinal layers.

During the next month the papilloedema gradually subsided. On March 1 visual acuity was: right, 6/18, with +2.25 D correctable to 6/6; left, 6/12 not improved with lenses. The haemorrhages were being absorbed on both sides and the swelling of the nerve heads had almost subsided.

On March 15 the patient complained of blurred vision with the left eye. Examination next day revealed a fresh macular haemorrhage. Papilloedema had subsided and the disks were pale.

On March 31 visual acuity was: right, 6/12, correctable to 6/6; left, 6/24 not correctable by lenses. Nearly all the haemorrhages originally seen had been absorbed and their previous sites were discovered with difficulty. The more recent left macular haemorrhage was being absorbed but was still evident, and the pallor of the disks was more pronounced on the right side than on the left.

COMMENT

The pathogenesis of papilloedema, as of most pathological types of oedema, is obscure. Watkins *et al* (1941) reported three cases of thrombocytopenic purpura and a case of anaemia due to bleeding gastric ulcer in all of which papilloedema was noted. In discussing the mechanism, they postulated a local reaction of the tissues of the optic nerves to anoxaemia. A general oedema of the brain due to the same mechanism is also mentioned as a possible factor. They did not enlarge on this, but presumably increased capillary permeability due to oxygen lack is responsible for the abnormal accumulation of extracellular fluid. It is not clear why this process should affect one optic nerve more than the other (in their fourth case the left showed elevation of 4 to 5 dioptres, the right only 1).

Simpson (1948) reported three cases of emphysema with papilloedema. He attributed the latter to a rise of cerebrospinal fluid pressure from cerebral vasodilatation due to oxygen lack and carbon dioxide accumulation in the arterial blood. It is possible that such a mechanism may operate in anaemia. However, these theories imply a threshold level of anoxaemia and cerebrospinal fluid pressure beyond which one would expect papilloedema to occur. Yet C.S.F. pressure may be high and many cases of severe anaemia may be seen without papilloedema being observed. Furthermore, the swelling may be much more prominent on one side than on the other. It would seem, therefore, that there must be some local factor the nature of which is not apparent.

This paper is submitted by permission of Mr. A. C. Turner, O.B.E., F.R.C.S.Ed., Chief Medical Officer, Anglo-Iranian Oil Company.

H. A. REID, M.B., M.R.C.P.Ed.
W. HARRIS, M.R.C.S., D.O.M.S.
Anglo-Iranian Oil Company.

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Reviews

DEVELOPMENT OF VISUAL RESPONSES

Vision. Its Development in Infant and Child. By Arnold Gesell, M.D., F. L. Ilg, M.D., and G. E. Bullis. Assisted by V. Ilg, O.D., and G. N. Getman, O.D. (Pp. 329; 72 figures. £2 2s.) London: Hamish Hamilton. 1950.

This book is unique in the literature and therefore must excite interest. Its genesis lies in the Yale Clinic of Child Development, which since 1911 has been concerned with the development of early human behaviour. By an elaborate series of carefully controlled observations—much of it in photociné records through one-way vision screens which leave the child and his attendants (when present) unembarrassed—the behaviour of the child and his reactions to various visual stimuli have been recorded and charted from birth at periodic intervals until school age. The study has even included the "foetal infant"—the prematurely born infant—from the age of 28 weeks after conception. The research includes an astonishing number of observations relating not only to visual responses but to the whole system of tonic and kinetic reflexes concerned in postural activities, in which the eyes are important agents. The study is a fascinating one for the physiologist, the paediatrician, the psychologist, and the ophthalmologist. The authors conclude that even in his ocular equipment the infant or child is not a miniature adult, but is qualitatively a different organism, constantly changing, and they hope that, if the techniques described are further elaborated, an appraisal and supervision of the efficiency and development of the visual (and other) functions should be possible from the earliest stages of life.

The book is interesting to read, but to appreciate it the reader must understand the American language. Moreover, he must understand that much of the exuberance—whereby the eye is described as the jewel of organic evolution and *Tarsius* as leaping around with suprasonic speed—is truly American in its delightful enthusiasm.

STEWART DUKE-ELDER.

RHEUMATIC DISEASES

Manual of Rheumatic Diseases. By W. P. Holbrook, M.D., and D. F. Hill, M.D. (Pp. 182; illustrated. $4.25.) Chicago: The Yearbook Publishers Inc. 1950.

This book is dedicated to the general practitioner and justifies the intentions of its authors. They do not attempt to present a documented scientific treatise or to cover all aspects of the subject, but to provide a practical guide to diagnosis and treatment, especially in home practice. They give a timely warning against waiting for miracle drugs and cures while neglecting the simple methods directed to preventing the development of crippling and deformity and raising resistance by attention to the general health. They describe a basic routine line of treatment which will have a psychological effect, improve the general health, and serve as a basis for such other methods as may be desirable. The authors hold that the most valuable physiotherapy is training the patient's own muscles through carefully directed exercises, thus maintaining muscle tone and range of joint movement and helping the circulation. They warn against excessive use of heat treatments, for while comforting at the time they tend to keep the disease active and do more harm than good in the long run.

Transfusion is often helpful, but vaccines and many other procedures which have been used in the past are condemned as mainly worthless. Gold treatment has been found beneficial in a large proportion of cases but demands careful supervision, signs of toxic effect being sought and frequent blood examinations made. Clear instructions for its use are given. A valuable feature of the book is the attention given to prevention of deformity, especially in home treatment. Tables of appropriate exercises are included, and the authors emphasize the importance of rest and avoidance of any exercise entailing over-use of inflamed or painful joints. They recognize the importance of trauma, postural and occupational strain, and general wear and tear in the aetiology of osteoarthritis. Excellent illustrations guide the reader in the use and application of splints, supports, crutches, and the like.

The authors discuss the most recent developments clearly but briefly. Cortisone and A.C.T.H., the adrenocorticotropic hormone, are fully described from their own practical experience. This is a book which the general practitioner will find most helpful and which the specialist may read with pleasure and profit.

C. W. BUCKLEY.

MEDICINE IN BULK

The Practice of Medicine. By J. C. Meakins, C.B.E., M.D., LL.D., D.Sc. Fifth edition. (Pp. 1,558. £4 15s.) London: Henry Kimpton. 1950.

Gravimetrically considered, this is probably the weightiest textbook of medicine yet published in one volume, and of its 1,500 pages over 1,000 come from the pen of Professor Jonathan Meakins himself. It is a monument to his erudition, to the range of his knowledge, and to his industry. Yet the reviewer cannot do otherwise than ask himself to whom this huge volume is addressed. Is it intended as a companion to the student's medical studies or is it meant as a work of reference for the practitioner? It is too long, too detailed, and too much concerned with uncommon diseases for the first; for the opposite reasons it cannot play the second role. Perhaps its handsome format and its wealth of illustrations may beguile the clinical tyro into expending £4 15s. on its purchase, but it is certain that no hard-driven student in his final year would make it the handbook for his qualifying examination. For all this, its success is proved by the demand for five editions in four years.

These observations are engendered by the increasing bulk of all the current textbooks of medicine, which reaches a critical level with the present work. There is need for an author who will write for students; who will limit himself to essentials and principles, seeking what he may omit rather than what he may include; and who will avoid the meretricious lure of "encyclopaedism." No textbook can take the place of the great systems of medicine, and those who require information on rare diseases will always need to consult original papers and monographs.

The individual sections in this book are excellent. Perhaps those on the respiratory and cardiovascular systems by Professor Meakins, and that on the nervous systems by the late Dr. Peterson, deserve particular mention. The introductory chapter entitled "An Introduction to the Practice of Medicine" reveals the broad view and

mature wisdom which mark the approach throughout the book. It is perhaps inevitable that the space devoted to some diseases should seem excessive and to others inadequate ; thus three pages are occupied by the section on acute diffuse lupus erythematosus, three-quarters of a page by the description of Bouchet's disease, while the common and important complaint glandular fever is allotted less than one page. Although it approaches the limits of portability, this book has many excellences : not the least of these are its numerous illustrations and its fine printing.

R. BODLEY SCOTT.

THE ELDERLY PROSTATE

Leitfaden für Prostatiker. By Professor J. Minder, M.D. (Pp. 68 ; 7 diagrams. M. 4.80.) Stuttgart: Georg Thieme. 1950.

Most British doctors would probably agree that the less a patient knows about his complaint the better. It is just because of our knowledge that we doctors as a class are sometimes so troublesome to those to whom we appeal for advice on our own poor bodies. But here is a book which deliberately sets out to tell every man what may happen to his prostate. Anything more likely to make him a prey of the quack it is difficult to imagine.

Yet for those for whom it is *not* intended, young doctors starting in practice, the book would be simply invaluable. The author gives a short and clear account of the anatomy of the prostate, of the mechanism of its enlargement, and of the way in which this may cause trouble. He draws a useful distinction between the troubles which age invariably brings and actual prostatic disease, and carefully explains the means by which this can be recognized. With clarity and precision he describes the pathological changes in the bladder which gradually develop, the means by which to some extent they may be avoided, and the later effects of back pressure on the ureters and kidneys.

There is an admirable chapter on instrumentation and especially on the art of passing a catheter. Whether a patient could be expected to take such precautions is questionable, but there is no doubt that a doctor should, and many a doctor would be grateful for the hints given here. The author gives a full account of medical methods of maintaining asepsis in the bladder and of combating sepsis, should it arise, with the most modern methods of drug therapy. Operative methods are naturally only referred to briefly, but every surgeon will agree with the remark that, whatever new methods may be introduced, "The best method is that in which the surgeon is most skilled and with which he has obtained the best results."

This little book is written in very simple German and with a clarity that is rare. It is full of really useful hints and will amply repay perusal.

HENRY SOUTTAR.

CHILDREN IN COURT

The Child and the Magistrate. By J. A. F. Watson. (Pp. 367. 12s. 6d.) London: Jonathan Cape. 1950.

Although Mr. Watson is not a doctor, his book can be safely recommended to any medical man who is clinically minded. This second edition of his well-known book, largely rewritten and brought up to date, must be studied as a textbook by every magistrate of a juvenile court, but it is written in such a way that anyone who has to do with children can thoroughly enjoy it from beginning to end. Those of us who do not exclude psychological cases from our out-patient clinics will recognize on every page the children with whom we are concerned, and it is very important for us to be able to see these same children through the eye, the human and kindly eye, of this magistrate.

If the book gives one qualms at all, it is by making one realize what a great deal depends, in the management of the delinquent who has arrived at the stage of court proceedings, on the clinical sense and the human understanding of the magistrates, and there cannot be many in whom are combined (as they are in Mr. Watson and his colleague Mr. Basil Henriques) a love of children with a knowledge of the law and of court procedure. Sentimentality will not do. The complexities of the Education Act, 1944, the Children Act of 1948, the Criminal Justice Act of 1948, and the Justices of the Peace Act of 1949 have to be known to the magistrate as anatomy and physiology have to be known to the doctor. Moreover, magistrates need to keep in mind that it is "the public interest which, as a court of justice, must always be their paramount consideration."

I think that general practitioners as well as psychiatrists should know what to expect of the juvenile court, and there can be no better way of getting this knowledge than through the enjoyment of this book. Incidentally, there are plenty of amusing tit-bits to help the reader along.

There is one thing which will come as new to many readers. There is a gap in management left by the serious attempt which is being made to eliminate corporal punishment. This gap is to be filled by attendance centres, provision for which is laid down in the Criminal Justice Act of 1948. However, only one exists as yet. Mr. Watson has great hopes from this new provision, but one feels that their success will depend not only on the way they are run but on their being used by the magistrates for the right kind of case—that is to say, for the child who is able to make use of a limited corrective.

D. W. WINNICOTT.

BOOKS RECEIVED

Review is not precluded by notice here of books recently received

Jealousy in Children. By E. Ziman, M.D. (Pp. 236. 12s. 6d.) London: Victor Gollancz. 1950.

First Aid to the Injured. Authorized Textbook of the St. John Ambulance Association. 40th ed. (Pp. 284. 3s.) London: The St. John Ambulance Association. 1950.

What is Hypnosis? By A. Salter. (Pp. 94. 8s. 6d.) London: Athenaeum Press. 1950.

Text-book of Orthopaedic Medicine: Treatment by Manipulation and Deep Massage. By J. Cyriax, M.D., B.Ch. Vol. 2. (Pp. 335. 18s. 6d.) London: Cassell. 1950.

Practical Gynecology. By W. J. Reich, M.D., F.A.C.S., F.I.C.S., and M. J. Nechtow, M.D. (Pp. 449. 80s.) London: J. B. Lippincott. 1950.

Surgery of the Shoulder. By A. F. DePalma, M.D. (Pp. 438. 140s.) London: J. B. Lippincott. 1950.

Surgery of Cataract. By D. B. Kirby, A.M., M.D., LL.D. (Pp. 695. 240s.) London: J. B. Lippincott. 1950.

A Companion to Manuals of Practical Anatomy. By E. B. Jamieson, M.D. 7th ed. (Pp. 736. 18s.) London: Geoffrey Cumberlege. 1950.

Practical Periodontia. By H. M. Robb, D.D.S., B.Sc. (Pp. 117. 21s.) London: Geoffrey Cumberlege. 1950.

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PROGNOSIS OF FOETAL ASPHYXIA

Doubts about the ultimate prognosis of severely asphyxiated infants at birth constantly assail those who are responsible for their resuscitation. Methods of obtaining information lack precision. The usual and less satisfactory one has been to correlate the clinical findings in a group of children, which is usually specially selected, with the birth history. Schreiber[1 2] inquired into the birth history of 900 patients seen by him because of defects such as cerebral palsy and mental deficiency and found a history of apnoea at birth in 70%. Faber[3] investigated 99 children with mental deficiency, spasticity, or convulsions—in all of whom cerebral atrophy was demonstrated—and discovered there had been prolonged apnoea or cyanosis at birth in 22. He thought that nitrous oxide anaesthesia, when given to the mother with less than 15% oxygen, carried with it a particularly grave risk of asphyxia to the foetus. Belnap and his colleagues,[4] examining 419 defective children aged 8 months to 14 years, ascribed 52% to birth injury and anoxia. Rosenfeld and Bradley[5] studied 673 children admitted to a psychiatric hospital for behaviour disorders in order to determine the effect of asphyxia in infancy on subsequent behaviour. There were 28 children with a history of difficult resuscitation at birth and 126 who had had severe whooping-cough. The authors found that the children with a history of asphyxia showed a characteristic syndrome of unpredictable variability in mood, hypermobility, impulsiveness, lack of attention, varying inability to recall material previously learned, and conspicuous difficulty with arithmetic. A syndrome consisting of at least five of these six traits occurred more than eight times as frequently as in the control group.

Preston[6] reviewed the prenatal and natal history of a group of children, some of whom were chosen for study because of difficulty at birth. This group, in which anoxia had occurred in 132 cases, was therefore a selected one. Those who had had lesser degrees of asphyxia were hyperactive and hyperexcitable; those in whom the asphyxia had been more severe were apathetic and readily lost emotional control. Convulsions occurred in 28% of all these patients. The intelligence quotient was within normal limits in 74%. The children tended to lack concentration, to be difficult to discipline, to play badly with others, and to be slow to learn from experience. Gesell and Amatruda[7] carried out a different form of investigation. In the course of their developmental examinations they picked out a group of infants with slight abnormalities in the pattern of movement, and with clumsiness and incoordination in finer manipulation. They were apt to have hyperactive reflexes, an exaggerated response to noise, early speech difficulties, and personality deviations. All recovered completely without sequelae. They ascribed this to minimal birth injury, arguing, quite rightly, that the history of normal delivery by no means excludes the possibility of birth injury and asphyxia.

A much more satisfactory method of observation is a longitudinal study, from birth onwards, of children who were known to have suffered asphyxia at birth. Darke[8] watched the development of 19 children who had severe asphyxia at birth and compared their I.Q. with that of siblings or parents. Their mean I.Q. was 88, and that of the controls was 100. Two of the 19 children had a higher I.Q. than the controls. Stevenson and Stuart[9] kept under observation 226 children who were in a poor physical condition at birth as a result of asphyxia, prolonged labour, prematurity, or maternal toxaemia. They found a highly significant relationship between this poor physical condition and childhood maladjustment. In animal experiments Windle and Becker[10] submitted pregnant guinea-pigs to the effect of asphyxia. The offspring showed not only various motor disorders but such deviations from normal as feeding difficulties and unusual quiescence: histological changes in the brain were demonstrated in 65% of the animals at necropsy. Different results were reported by Keith and Norval,[11] who followed 216 children born after prolonged labour. One hundred and eleven had asphyxia livida or pallida, or delayed respiration for more than a minute. There were more deaths among the asphyxiated infants in the newborn period than among the controls, but those who survived developed normally over a four-year period. Even severe asphyxia with delay of several minutes in the onset of respiration did not seem to have any effect on the survivors. The same authors described their results in more detail

[1] *J. Amer. med. Ass.*, 1938, **111**, 1263.
[2] *J. Pediat.*, 1940, **16**, 297.
[3] Ibid., 1950, **37**, 326.
[4] *Amer. J. Dis. Child.*, 1947, **4**, 1.
[5] *Pediatrics*, 1948, **2**, 74.
[6] *J. Pediat.*, 1945, **26**, 353.
[7] *Developmental Diagnosis*, 1947, New York, p. 215.
[8] *J. Pediat.*, 1944, **24**, 148.
[9] *Amer. J. Dis. Child.*, 1950, **79**, 931.
[10] *Amer. J. Obstet. Gynec.*, 1943, **45**, 183.
[11] *Proc. Mayo Clin.*, 1950, **25**, 11.
[12] *Pediatrics*, 1950, **6**, 229.
[13] *Amer. J. Obstet. Gynec.*, 1941, **42**, 686.
[14] *Arch. Dis. Childh.*, 1950, **25**, 351.
[15] *Amer. J. Obstet. Gynec.*, 1931, **21**, 537.

in another paper.[12] Twenty-four out of 25 full-term babies who began breathing after a delay of 1–5 minutes, 15 out of 16 who began after 6–10 minutes, and all five who began after 11–15 minutes were normal at the age of 1 year and remained so. Similarly, McPhail and Hall,[13] who tested the intelligence and behaviour of 34 children of school age who had been apnoeic at birth, found no abnormalities among them.

Finally an informative report has just appeared from Belfast, where Campbell, Cheeseman, and Kilpatrick[14] examined the records of the Royal Maternity Hospital in that city for the years 1938–41, and selected for study all the infants who had been born in a condition of asphyxia pallida or asphyxia livida lasting for a stated period of over two minutes. In the end they were able to trace 61 out of 89 "asphyxiated" infants and to compare them with a control group of 134 infants born at the same time. The examination of the children was so arranged that the clinicians did not know from which group a child came. The authors found no significant differences in the average physical measurements of the two groups of children, in haemoglobin levels, or in intelligence (assessed from Raven's matrices). They conclude that the results of their investigation do not support the hypothesis that asphyxia neonatorum is a common cause of later physical or mental retardation.

There are great difficulties in the interpretation of these reports. It seems clear that those who are consulted about defective children are apt to obtain a too pessimistic view of the outcome of foetal asphyxia. There can be little doubt that asphyxia can and does cause spasticity, convulsions, and mental deficiency, but it is not clear how often it does so; and it is still less clear whether it leads to behaviour disorders. A mentally or physically defective child is more likely to develop psychological disorders than other children, and any study designed to determine the relation of asphyxia at birth to subsequent behaviour disorders should exclude such defective children. A special difficulty is the absence of a yardstick by which to measure behaviour disorders. Behaviour which some would consider abnormal, others, with greater knowledge of normal child psychology, would consider normal. A child's behaviour depends largely on his inherited character and on environment, in particular the parental attitudes and management, and this might be related to the circumstances of his birth. Elderly primiparae are more likely to have obstetrical difficulties than others, and the emotional background of an only child of elderly parents, particularly if there were severe obstetrical difficulties or other factors which might render another pregnancy impossible, could well lead to overprotection and behaviour disorders. A further possibility is that a child may be cyanotic at birth because of cerebral agenesis, with its widespread effects on the nervous system, so that the subsequent defects need not be the result of the asphyxia at all.

The answer to some of these problems can come only from the study of larger series of cases, but here again there are difficulties. The higher the standard of obstetrics the lower is the incidence of foetal asphyxia.[7] Darke could find only 49 cases of severe asphyxia out of 25,261 deliveries, and only 19 of these could be traced. The investigation, which must be adequately controlled, should include a standard method of measuring the degree of asphyxia, such as that of Flagg.[15] The development of the infant should be assessed by Gesell's standards and by the other accepted methods at later ages. The psychological examination should be undertaken by a physician or psychologist who is thoroughly conversant with normal child psychology and who does not know the birth history of the children whom he is examining. The results of such studies might do much to lessen the widespread pessimism about the outlook for these children, and they would certainly help doctors to give a sounder opinion to the parents.

REGULATION OF BODY TEMPERATURE

The war gave great impetus to research on the relation of man to his working environment, and many of the observations have added to our store of biological knowledge. Much attention has been given to the effects of changes in the environmental temperature on the body temperature, and Glaser[1 2] has recently reported the results of some of this work. When lightly clad men were subjected to an environmental temperature of 0° C. for 1½ hours the rectal and skin temperatures decreased. The rectal temperature fell steadily to about 1° C. below the initial level. As might be expected, the reduction in skin temperature, as measured on the forehead and on the hand, was much more marked, though the course of the temperature change and the amount of the fall were different in the two skin areas. The forehead temperature decreased rapidly during the first 15 minutes, then more slowly, until after 30 minutes it remained fairly constant at an average of 4° C. below the original reading. The hand temperature, however, did not reach a steady value but fell progressively by about 11° C. After the 1½-hour period of cooling, the subjects were allowed either to rest at room temperature (17° C.), to do alternate periods of exercise and rest at room temperature, or to rest in a warm room (30° C.). Again the temperature

pattern varied between the different regions. The rectal temperature, surprisingly, continued to fall for about 15 minutes and then gradually rose over a period of one hour, when it became stable. The level at which it became stable, however, was always below the reading taken before cooling, and, in the case of those subjects who had rested at room temperature, was actually below the last reading in the cold. On the other hand, the forehead and the hand temperatures immediately began to increase rapidly, and within 1¼ hours settled down either distinctly above the first observation of the day if the subject sat in a warm environment, or regained the pre-experimental level if the subject remained at room temperature.

In general the results show that the rectal and skin temperatures become fairly constant after about 1¼ hours' warming. At this point it is presumed that a balance has been struck between heat production and heat loss, though the body temperature as measured in the rectum was not restored. This seems to indicate that the establishment of thermal balance takes precedence over the maintenance of a constant body temperature. If this is generally true, then perhaps the statement that man has efficient mechanisms to maintain a constant temperature should be modified to read that he has efficient mechanisms for the maintenance of thermal balance. It is interesting to note that all of Glaser's subjects felt comfortable within 30 minutes of leaving the cold—some time before heat balance had been achieved—and they often had no sensation of cold even with a rectal temperature lower than that at which they had shivered or felt cold before. It is thus apparent that the sensation of warmth must be differentiated from the total heat content of the body. For instance, a person may feel warm when the body temperature is falling, provided that the skin temperature is such as to arouse a sensation of warmth in the skin, and, in reverse, a person in thermal balance may feel cold because of a low skin temperature. Glaser therefore concludes that a satisfactory state of body temperature depends on balance or near balance between heat production and heat loss and a range of skin temperature which is subjectively comfortable. He found no relationship between rectal temperature and shivering or sweating. Shivering did not appear to depend on a fall of the rectal or the skin temperatures to any particular level. It usually began ¾–1 hour after cooling had commenced, and its onset slowed down the fall of rectal temperature or even caused a slight rise, due no doubt to increased metabolism. The rectal temperature at which shivering began varied from subject to subject and in any one individual from time to time. As Glaser points out, a relationship between rectal temperature and shivering appears highly improbable, since there is no evidence that receptors which initiate shivering lie in the wall of the rectum. The sweating that occurred during warming was not related to the attainment of any particular rectal temperature: sometimes it occurred at a lower rectal temperature than that at which the subject began to shiver on the same day.

The unexpected finding that rectal temperature falls initially during warming may be due to the peripheral vasodilatation that occurs immediately on entering a warm environment, increasing heat loss at the expense of deeper tissues. Another suggestion made by Glaser appears unlikely—that the temperature in the rectum may be affected by the temperature or quantity of blood returning from the lower limbs through the haemorrhoidal venous plexus to the inferior mesenteric vein. The only connexions described are anastomoses between the veins of the buttock and the rectal veins. The rest of the blood returning from the lower limbs could perhaps affect the temperature of the blood in the rectum indirectly by cooling the blood in the iliac arteries. Glaser also points out that various parts of the body may change their temperature within comparatively wide limits, while the body as a whole remains in a state of thermal balance or achieves a balance at a new level, and he suggests that the balance is maintained by the appropriate movement of blood between the periphery and the internal organs. Previous work[3-5] has shown that the reduction of peripheral blood flow which is a consequence of cooling is accompanied by an increased intra-abdominal and cerebral blood flow, and that the opposite happens as a result of warming. It has also been suggested that blood may be diverted from the periphery to the lungs and vice versa during the redistribution process.[6] In another paper Glaser[7] has demonstrated that in a cold environment the vital capacity of the lungs is decreased as well as the volume of the arm, and in a hot environment both are increased. It seems reasonable to suppose that the cooling resulted in "movement" of blood from the limbs to the lungs, so reducing the vital capacity, while warming caused movement in the reverse direction with an increase in the vital capacity. Further evidence of this redistribution has been given by Grayson,[8] who has shown, as a result of a study of the temperature of the intestine exposed by colostomy or ileostomy, that within certain limits cooling causes an increased intestinal blood flow as well as a reduced peripheral blood flow and that warming has the opposite effect.

[1] *J. Physiol., Lond.* 1949, **109**, 366.
[2] *Nature, Lond.*, 1950, **166**, 1068.
[3] Müller, O., *Dtsch. Arch. klin. Med.*, 1905, **82**, 547.
[4] —— and Siebeck, R., *Z. exp. Path. Ther.*, 1907, **4**, 57.
[5] —— and Veiel, E., *Samml. klin. Vortr.*, 1909–10 (*Innere Med.* 194–196), p. 641.
[6] Sjöstrand, T., *Skand. Arch. Physiol.*, 1935, **71**, *Suppl.*
[7] *J. Physiol. Lond.*, 1949, **109**, 421.
[8] Ibid., 1949, **109**, 439.

CHOREA

It cannot be said that we know much more about chorea than did our grandfathers. It remains a puzzle that a disease apparently due to a profound disturbance of the central nervous system should fail to show histological changes in the nervous system and should leave no permanent neurological defects. Nor is the nature of the connexion between chorea and rheumatism yet understood. A recent study from Scandinavia seeks once more to shed light on this. Lewis-Jonsson[1] surveyed all cases of chorea known to have occurred over a 35-year period from 1910 to 1944 in a county in the south of Sweden. Out of 704 choreic patients whose fate was partly known there were 467 cases which at one time or another showed rheumatic signs—polyarthritis, carditis, erythema annulare, or nodules. In 22 cases the chorea was deemed secondary to another disease such as hemiplegia, cerebral neoplasm, encephalitis, or Huntington's familial chorea. There remained 215 cases of cryptogenetic chorea.

Analysis of the 467 cases of rheumatic chorea gave similar results to those reported by other authors: females were more than twice as common as males; the age of the youngest patient was 3 years, and most cases came within the 6–14 years age group; the onset was more often between November and May than at other times of the year, the seasonal incidence following a similar trend to that of rheumatic fever in the same region. In the group of patients with chorea of unexplained origin the seasonal incidence and the age of those affected were much the same as in the group of rheumatic choreics, and Lewis-Jonsson concludes that most of these cases are due to monosymptomatic rheumatic infection. This view is supported by the fact that the proportion of those who are found to develop rheumatism rises as the period of observation is lengthened. Thus Sutton and Dodge[2] found that, of 91 cases of initially uncomplicated chorea, heart disease was present in 10% after 3 years, in 15% after 6 years, and in 40% after 10 years. Nevertheless there remains a large proportion of patients, probably over 50%, who never develop any other form of rheumatism.

If a patient has chorea with no past or present evidence of rheumatism, is it possible to tell whether or not he will eventually develop rheumatism? According to Kagan and his colleagues[3 4] the erythrocyte sedimentation rate enables this to be done, for after observing 165 patients with chorea they concluded that a normal E.S.R. during a first attack indicated that rheumatism was not the cause. However, this conclusion is at variance with that of Lewis-Jonsson, who found a normal E.S.R. in nearly half of a series of 150 cases of rheumatic chorea. The experience of most clinicians is that if the E.S.R. is raised in chorea then some other rheumatic sign, such as carditis, is usually present; but that in uncomplicated chorea, even if the patient later develops rheumatism, the E.S.R. is usually normal. Evidently the factor in rheumatic infection which causes a raised E.S.R. is not the same as that which gives rise to choreic symptoms. Chorea is a well-recognized complication of pregnancy. These patients are older than the average choreic, and the disorder tends to take a severe form, but in other respects it does not differ from ordinary chorea. In particular, rheumatism is equally common, and in 7 out of 18 pregnant choreic patients in Lewis-Jonsson's series rheumatic infection had preceded the chorea. The part played in chorea by emotional disturbance is another problematic feature. This is present to some degree in most cases, while in a minority the disease seems to have followed directly upon some mental trauma. This occurred in 59 of the 215 cases of rheumatic chorea in Lewis-Jonsson's series. As an alternative to this author's view that uncomplicated chorea is monosymptomatic rheumatism, chorea might perhaps be regarded as a psychosomatic symptom which rheumatic subjects are peculiarly liable to develop and which can be provoked by a variety of stresses—emotional, metabolic, or infective, the commonest infective factor being rheumatic fever itself.

The facts known about this disease provide some basis for its treatment. Clinical evidence of active rheumatism, such as carditis, will of course mean that the patient needs rest in bed: in its absence strict physical rest will not necessarily be indicated, though hospital care, with or without full bed rest, may well have a part to play in helping the child to overcome its disorder. It is difficult for the patient's family to maintain that healthy neglect of the main symptom which may be an essential part of treatment. Mild sedation apart, drugs have no part in treatment, while the little use that is now made of pyrotherapy—whether by the use of protein shock, "nirvanol," or the "hot-box"—indicates the ineffectiveness of such methods.

[1] *Acta paediatr., Stockh.*, 1949, *Suppl.* 76.
[2] *Amer. J. med. Sci.*, 1938, **195**, 656.
[3] *J. Pediat.*, 1947, **31**, 322.
[4] *Amer. J. Dis. Child.*, 1949, **78**, 306.

FATAL INTESTINAL OBSTRUCTION

There has been a steady fall in the mortality rate of intestinal obstruction treated surgically. In one series of cases in which the obstruction was uncomplicated by strangulation or carcinoma the mortality was as low as 1%,[1] but in another series in which gangrenous bowel was found at operation over half the patients died.[2] Nemir and his colleagues[3] have tried to find out why the presence of strangulated gut should have such an adverse effect. By surgical means they induced total obstruction in dogs, with strangulation of part of the small intestine. After the animals had recovered from the anaesthesia it was possible to give continuous intravenous infusions as they walked about the cage. Two different types of fluid were found to be extruded by the strangulated gut. A pink fluid came off the peritoneal surface; large quantities might collect within the peritoneal cavity, and death occurred from oligaemic shock. When this loss of fluid was counteracted by intravenous infusion, death was postponed for only a few hours. The animals died suddenly soon after the first appearance in the blood of another pigment which was also found in the black fluid secreted within the lumen of the strangulated gut. It appeared that as soon

[1] Eliason, E. L., and Welty, R. F., *Ann. Surg.*, 1947, **125**, 57.
[2] Schlicke, C. P., Bargen, J. A., and Dixon, C. F., *J. Amer. med. Ass.*, 1940, **115**, 1411.
[3] *Ann. Surg.*, 1949, **130**, 857, 874.

as this fluid left the gut and was absorbed into the general circulation, where it could be identified spectroscopically, the animal collapsed and died. Separate experiments showed that the pink fluid from the external surface of the gut was non-toxic to normal dogs, whereas the dark fluid was very toxic. The authors point out that the black pigment is useful only for identifying this particular exudate, and they do not assume that the pigment itself is toxic. Although cultures were made and were positive for a number of organisms, they do not discuss the possibility that the factor responsible for death might be bacterial in origin. It would be interesting to repeat the experiments in dogs that have been given large quantities of penicillin. Although the toxic factor remains unidentified, its source seems to have been established. Further support is given—if, indeed, any is needed—for early surgery, but it also behoves the surgeon to handle the strangulated gut with care to prevent any further dissemination of its poisonous contents.

A.C.T.H. TREATMENT OF ACUTE RHEUMATISM

The interest aroused by the successful treatment of rheumatoid arthritis with cortisone and pituitary adrenocorticotropic hormone (A.C.T.H.) has perhaps rather overshadowed the claim first made by Hench, Kendall, Slocumb, and Polley[1] that cortisone also favourably influenced acute rheumatism. If some method of treatment could be found which effectively arrested the course of acute rheumatism and rheumatic heart disease it would be not only of profound interest in the study of the disease but of inestimable value in eliminating an important cause of death among young people. A valuable report on the effect of A.C.T.H on rheumatic fever and rheumatic carditis has come from Massell and his colleagues[2] in Boston. They studied 10 patients aged from 7 to 21 who between them showed most of the clinical manifestations of rheumatic fever. The first case reported is perhaps the most impressive. This was a girl, aged 11, admitted to hospital about a month after the onset of acute polyarthritis. She was severely ill, with a pericardial friction rub and extensive subcutaneous nodules—in fact, she belonged to that group of patients who are nearly always left on recovery with a severe cardiac lesion. She received A.C.T.H for 51 days in doses diminishing from 36 mg. to 2 mg. daily. Fever and joint pain disappeared in 24 hours and the pericardial friction in three days. Mitral and aortic murmurs could then be heard. Improvement, however, continued, and at the end of the treatment her heart was normal clinically, electrocardiographically, and radiologically. In the other cases the response was less dramatic. Reporting on the results obtained with cortisone or A.C.T.H. in 11 cases of acute rheumatic fever, Hench and his colleagues[3] state that it will take months or years to determine whether these hormones will prevent the initial development or aggravation of rheumatic carditis.

It is important to remember that patients with rheumatic fever as ill as the first child can make, though rarely, a complete clinical recovery. From these 10 cases it is clear that A.C.T.H. will control the fever and the arthritis of acute rheumatism in 24–48 hours, but so also will salicylates. The difficulty is notorious of assessing the effect of any form of treatment in acute rheumatism, which in different patients runs such a varying course. The only patient with chorea treated by Massell and his colleagues improved immediately, but choreic movements persisted for 30 days. Subcutaneous nodules were observed in three children under treatment; they remained present for 45 and 50 days in two and were still present at the time the report was written in the third. The sedimentation rate, too, remained raised for anything from 6 to 87 days. On withdrawal of the drug there was a return in some cases of fever and joint pain, which cleared up again when an increased dose of the drug was given. This phenomenon is of course well known during salicylate treatment. The tendency of A.C.T.H. to cause retention of fluids sometimes led to an exacerbation of congestive heart failure; this, however, could be controlled by diuretics and restricted salt intake. Side-effects of the hormone treatment included the development of acne in six cases, roundness of the face in six cases, and striae in two.

From these studies it appears that stimulation of the adrenal cortex by A.C.T.H. can certainly benefit acute rheumatism, though of course this does not necessarily mean that an endocrine defect is concerned in the aetiology. It is not clear from the report of the Boston workers whether A.C.T.H. arrests the rheumatic process or whether it merely controls some of the clinical manifestations of the disease. Many consider that the latter is all that can be claimed for salicylates. While the effect of A.C.T.H. on acute rheumatism is of very great importance in the study of the disease, it may in the end prove to be comparable to that of salicylates.

[1] *Proc. Mayo Clin.*, 1949, **24**, 277.
[2] *New Engl. J. Med.*, 1950, **242**, 641 and 692.
[3] *Arch. intern. Med.*, 1950, **85**, 545.

SMALLPOX EPIDEMIC

Eleven cases of smallpox, including one death, had been reported in the Brighton area up to January 1 (6 p.m.) (see page 48). The usual precautions are being taken to stop the spread of the disease, but cases may appear elsewhere, especially among people who have been in direct or indirect contact with the R.A.F. officer who introduced the infection from Karachi. In December this man made three train journeys between R.A.F. stations in Scotland and Brighton. Doctors throughout Britain should therefore bear in mind the possibility that smallpox may occur in patients unaware of any contact with a suspected case. Notable features of this epidemic are the lack of diagnostic signs and symptoms in the initial case, with consequent late diagnosis, and the spread of infection to laundry workers by means of contaminated clothing.

We record with regret the death on December 30 of Mr. G. R. Girdlestone, who had an international reputation as an orthopaedic surgeon and was Nuffield Professor of Orthopaedic Surgery in the University of Oxford from 1937 to 1940. He was 69 years of age.

REFRESHER COURSE FOR GENERAL PRACTITIONERS

FUNGUS INFECTIONS OF THE SKIN

BY

F. RAY BETTLEY, T.D., M.D., F.R.C.P.

Physician for Diseases of the Skin, Middlesex Hospital, London

Most fungus infections of the skin involve only the epidermis and its appendages, the hair and the nails, with comparatively little reaction of the underlying tissues. For this reason they are not a very potent cause of serious disability except when complications arise. Their increasing prevalence, however, gives this group of infections importance. I propose to deal with them in roughly their order of frequency in this country at the present time.

Tinea Pedis

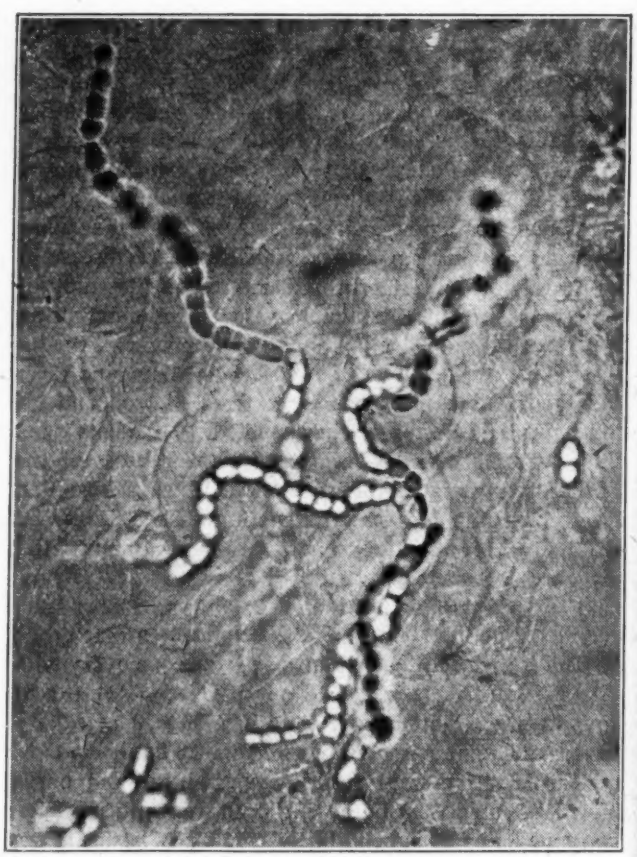
This occurs in two very different clinical forms, which are, however, often associated together either simultaneously or at different times in the same patient. The vesicular form nearly always starts in the centre of the sole, under the arch of the foot, as a group of perhaps half a dozen watery vesicles of large pin-head size which after a few days become superficial pustules. At this stage irritation may be considerable, but the thickness of the corneous layer is usually sufficient to keep the vesicles intact in spite of scratching.

The pustules gradually dry up and, as they do so, take on a brownish colour so that they are eventually shed in the form of thick brown scales a quarter of an inch or so across. It is a week or ten days before these scales are formed, and by this time fresh vesicles are often forming near by. This process may continue for some weeks, eventually appearing on the sides of the feet and toes and even on the dorsum of the foot. Even without treatment, however, the process eventually seems to burn itself out and the attack subsides for a time.

The interdigital form develops more insidiously and may consist of no more than a maceration of the skin between the fourth and fifth toes, which the patient attributes to moisture. The eruption is usually a little more extensive, affecting the under surfaces of the fourth and fifth toes and the skin between the third and fourth toes as well. Thick white sodden scales are formed, and, with any unaccustomed walking, painful fissures are likely to appear between and under the affected toes. Once this interdigital form of ringworm starts it seems to continue almost indefinitely, and probably forms a reservoir of infection from which attacks of vesicular ringworm on the sole periodically originate.

A generation or so ago this kind of ringworm was chiefly to be found in the Tropics and was spread in this country in the public schools and sports clubs. It has now become so widespread that the name "athlete's foot" is no longer merited. Dr. Jacqueline Walker has recorded that National Service men, on entry to the Army, showed a 3.8% incidence of foot ringworm.

The diagnosis is not always easy, and I believe that the clinical appearances of both types of the disease may be mimicked by other affections. For this reason it is most desirable to examine scrapings before treatment is started so that the diagnosis may be assured. All too often this examination is left until a preliminary period of treatment has failed, and the fungus may then be very difficult to find even though it was originally present and the cause of the trouble. For this examination a few scales or the roof of a vesicle are removed from the margin of the affected area, placed on a microscope slide, and flooded with 10% liquor potassae. After five or ten minutes the preparation is sufficiently softened to flatten out under pressure of the coverslip and is then



Skin scales showing fungus: unstained preparation, high power. From a case at the Middlesex Hospital.

examined unstained with the high-power objective. In an untreated case, fungus is usually easy to find after two or three minutes' search (see Figure); if it is not found quickly it is better to take fresh scrapings rather than to spend a long time over any individual slide.

Complications

It is the complications of foot ringworm far more than the disease itself which cause incapacity. They are

nearly always due to secondary infection with pyococci or to contact dermatitis resulting from the prolonged use of antiseptics and fungicides.

Treatment

In my experience, the old-fashioned Whitfield's ointment is still as good as anything. Now that coco-nut oil is again available the original formula can be used :

℞

Salicylic acid, 15 gr. (1 g.)
Benzoic acid, 25 gr. (1.65 g.)
Coco-nut oil, 6 drachms (21 ml.)
Soft paraffin to one ounce (30 g.)

The ointment should be liberally applied twice daily and the foot enclosed in a thin cotton sock or a single layer of calico held in place by normal socks or stockings.

In recent years a good deal of research has been carried out into fungicides, and several elegant preparations are now on the market. There can be no doubt that most of them are very effective, perhaps more so than Whitfield's ointment, but most of them are more liable to cause contact sensitivity and a severe form of dermatitis which may be far worse than the original disease.

Three or four weeks' treatment is usually sufficient, and during this period the patient must be schooled to the regimen which he should adopt if he is to avoid recurrence. Recurrences are, of course, all too frequent. In some cases they seem to be definitely related to reinfection by walking bare-foot in a club changing-room or hotel bathroom. It is known that the organism may remain for months on an infected floor and that it may survive the ordinary process of washing with soap and warm water. Once the patient has shown that he is susceptible to this infection it is important that he should get into the habit of never putting the bare foot to the floor even in his own home. Socks should be sterilized either in the autoclave or by soaking for twelve hours in 3% cresol before they are washed. This period of sock sterilization should continue for a week or so after the skin has returned to normal. Bedroom slippers in which bare feet are placed should be either thrown away or packed with formalin swabs and enclosed in an airtight box for three days. Shoes which are worn with socks need not be dealt with.

Sometimes there is no history of reinfection, and in these cases attacks often seem to follow unaccustomed exercise in which the feet have perspired more than usual. There is little doubt that excessive perspiration predisposes to infection, and it is impossible to say whether the organism has been lying dormant in the skin during months of quiescence or whether there is, in addition, a fresh infection from without.

It is therefore important for the patient to avoid those things which are apt to lead to excessive perspiration of the feet. He should wear light shoes, avoiding rubber soles, and in summer open sandals are helpful. Thin cashmere socks are usually best. The feet should be washed every day with soap and water, swabbed with industrial spirit, and powdered with zinc oxide and starch powder to which 2% of boric acid may be added. When recurrence takes place the patient should be carefully questioned in order to ascertain, if possible, why the relapse occurred. A search should also be made for ringworm infection elsewhere on the skin, particularly in the nails.

Tinea Unguium

It is my impression that ringworm of the nails has become a good deal more common in recent years. Clinically, the most constant physical sign is separation of the distal part of the nail from the nail bed with a dirty yellow or orange discoloration. The nail usually becomes brittle, and may be ridged or furrowed. These changes are, of course, produced by the fungus growing in the substance of the nail, the nail having been originally well formed. For this reason the changes of ringworm are usually confined to the distal half of the nail and the proximal part remains normal. This is perhaps the most useful differential diagnostic point, since most other affections of the nails are true dystrophies and affect the nail matrix so that the irregularity of the nail is present from its first formation. Another useful diagnostic point is the fact that ringworm of the nail nearly always starts in one finger or toe and others are affected one by one at random. True dystrophies often affect all the nails simultaneously or, if they do not, are at least symmetrical. Ringworm of the nail produces no inflammatory reaction in the soft tissues around and no symptoms other than the ugly appearance and awkwardness resulting from brittleness and discoloration. For this reason the patient will usually complain of ringworm in a finger-nail, but an infected toe-nail may go unheeded, particularly if it is one of the smaller toes.

Ringworm of the nails is caused by the same organisms as those which may give rise to ringworm on the feet and elsewhere on the skin. An infected nail may therefore constitute a reservoir of infection from which tinea of the foot or groin may repeatedly develop.

Although the diagnosis can often be made with reasonable certainty on clinical grounds alone, it is essential to make sure by finding the fungus before embarking on the long and tedious treatment which is necessary. Clippings of nail and the debris beneath are steeped in 20% liquor potassae until macerated and examined in the same way as scales. The fungus may be difficult to find, and repeated careful search may be necessary.

Treatment

Sir Norman Walker referred to ringworm of the nails as "a calamity which no decent person would wish on his worst enemy." Treatment is difficult and exacting, and, to most patients, seems at first out of all proportion to the severity of the disease. It must be pointed out, however, and the patient must understand, that with half-measures the affected nails will never clear up ; they will remain a source of infection for the rest of the skin, and, as the years pass, more finger- and toe-nails are likely to be involved. The great difficulty in treatment arises because the fungus grows in the substance of the nail, the solid tissues of which protect the micro-organism from fungicides. *X*-ray treatment is without effect. The problem, therefore, is to remove the infected nail and thus to expose the fungus to attack with antiseptics.

Conservative treatment consists in cutting away the separated nail, as far as possible, with pointed scissors. The nail plate must then be systematically scraped as thin as can be borne ; the best instrument for the purpose is a piece of broken glass of suitable size, and the

scraping should occupy at least ten minutes every day. After the patient has scraped away as much as he can bear, a suitable application is 1:2,000 perchloride of mercury in 50% spirit. This treatment needs to be continued for two or three months. It is a treatment which requires a great deal of determination and perseverance on the part of the patient. It is seldom possible to carry it out properly on any toe-nail except that of the big toe, and, since it is time-consuming, it is not likely to be successful if more than one or two nails are affected. For these reasons I advise conservative treatment with any confidence only for an intelligent adult with one or two finger-nails affected.

The alternative is removal of the affected nails. In this way the bulk of the inaccessible fungus is removed, but it must be remembered that the organism remains on the nail bed and will infect the new nail if it is not dealt with. The affected nails are therefore avulsed under local or general anaesthesia and the nail bed lightly curetted to remove debris. A dressing of tulle gras may then be applied and need not be changed for a few days. After four or five days the tenderness of the nail bed rapidly diminishes and the area may then be dressed with Whitfield's ointment twice daily. In the case of toe-nails, rest in bed will be necessary for a few days, but normal shoes can usually be worn after a week or so. Fingers must be kept covered with a finger-stall, preferably of leather : it is not satisfactory for a rubber glove or finger-stall to be worn for long periods. Treatment with Whitfield's ointment should continue for two or three months, by which time the new nail will be growing up.

Even this drastic treatment, with its upset of the normal life of the patient, is not always successful, and it should be explained to the patient at the outset that, although this method is far more satisfactory than conservative treatment, there is nevertheless a substantial risk of recurrence. If this occurs the more drastic step of excision of the nail matrix may be considered. On the toes, particularly the small toes, this involves little disfigurement, but many patients do not like the idea. However, the permanent loss of toe-nails constitutes no disability, and excision of the nail bed is probably wise in recurrent tinea of toe-nails if finger-nails are not already affected. Permanent removal of finger-nails constitutes as much disfigurement as tinea and some degree of disability as well. For this reason I would advise repeated avulsion of finger-nails, if necessary, rather than complete excision.

Tinea Tonsurans

Had this article been written four or five years ago scalp ringworm would have taken first place. It became very common during the war, but, as soon as more normal conditions were restored and treatment could be better organized, the disease was brought under control. It is now comparatively uncommon. The clinical problem has been epitomized by Darier : "Any multiple scaly patches or dry eczema localized to the scalp in children impose the need for a meticulous search for broken hairs. . . . "

This type of ringworm is very seldom seen in adolescents or adults and affects principally children, especially boys between the ages of 5 and 10. The well-outlined scaly patches are not truly bald, since the hairs do not fall out by the roots but snap off, leaving the affected area covered with stumps. While this is the characteristic picture, some patients present no more than a scaling patch and the hairs do not seem to become brittle. In nearly all types of scalp ringworm the affected hairs fluoresce in ultra-violet light; this is best seen with a mercury vapour lamp screened with Wood's glass, which cuts off most of the visible rays. When this is shone on an affected scalp in a darkened room individual hairs containing the fungus shine conspicuously with a green fluorescence. In this way an infected child can be picked out in a few moments' examination, and even single infected hairs can be found at a stage when the disease could not be clinically diagnosed. For this reason a lamp to provide Wood's light is essential for the examination of contacts, the prevention of epidemics, and the detection of relapses. The fluorescence produced by soft paraffin should be carefully distinguished ; it is of a blue colour, and of course can be removed by cleaning the hair with ether.

Characteristic fluorescence with Wood's light is pathognomonic. In cases of doubt, or if Wood's light is not available, the diagnosis of ringworm should be confirmed by examining a hair in the same way as scales. The fungus appears as a close mosaic of regular rounded spores encircling the hair-shaft. A proportion of cases of scalp ringworm are due to the organism of cat or dog ringworm. Unlike ringworm of the human type, these cases seldom occur in epidemics, since the infection does not readily pass from one human to another. The distinction can be made with certainty only by culturing the organism.

Treatment

The treatment of ringworm of the scalp presents a problem similar to that of ringworm of the nails. The organism is protected by the relatively impervious material—in this case the hair-shaft—in which it grows. The search for a fungicide and for a vehicle which would penetrate into the hair-shaft was being actively pursued in this country a year or two ago. Now, however, the disease has become comparatively rare, and it seems as though these researches are likely to come to a standstill. Since, up to now, no certain way of reaching the fungus has been found, successful treatment depends on the removal of the infected hairs. They cannot be removed manually, since they are brittle and break off, and the only successful method which has stood the test of time is epilation with *x* rays. When carried out by an experienced operator this procedure is absolutely safe. The hair usually falls from the whole of the scalp about a couple of weeks after the *x* rays have been given and gradually grows again after a further lapse of three or four weeks. *X* rays have no effect on the fungus itself, and during the period of baldness Whitfield's ointment should be rubbed into the whole of the scalp twice daily.

This may seem a drastic treatment, but parents should be urged to adopt it without delay ; conservative treatment is exacting, time-consuming, and most uncertain in its results.

A child with scalp ringworm should, so far as possible, be isolated from other children until after *x*-ray epilation is complete. Throughout this period he should wear a washable linen cap in order to minimize the spread of infection. The detection of a case of ringworm imposes

the need of routine examination of his brothers and sisters, playmates, and schoolfellows. For this purpose Wood's light is indispensable.

Tinea Cruris

This is usually caused by organisms which may also infect the feet or the nails. Known as dhobie itch, it was formerly chiefly familiar as a tropical infection contracted by wearing clothing which had been infected during laundering. The eruption appears as a bright red itchy disk on the inner aspect of the thigh two or three inches below the crutch. The affected area is scaly and sometimes there are a few tiny vesicles at the margin; very often the margin is a little raised and palpable, with a tendency for the patch to clear up spontaneously in the centre. As the patch enlarges it extends to the inguinal region, thigh, perineum, or scrotum. In differential diagnosis this form of ringworm is to be distinguished from intertrigo; the latter always starts exactly in the groin fold and not a few inches down on the thigh. Extending from the fold, it usually affects opposing surfaces symmetrically. At the margin of a patch of intertrigo follicular lesions are very often to be seen: tinea of the thigh is never follicular. Unless the clinical diagnosis is quite certain it is always best to remove a scale, preferably from the margin of the patch, and examine it microscopically. The fungus is easy to find.

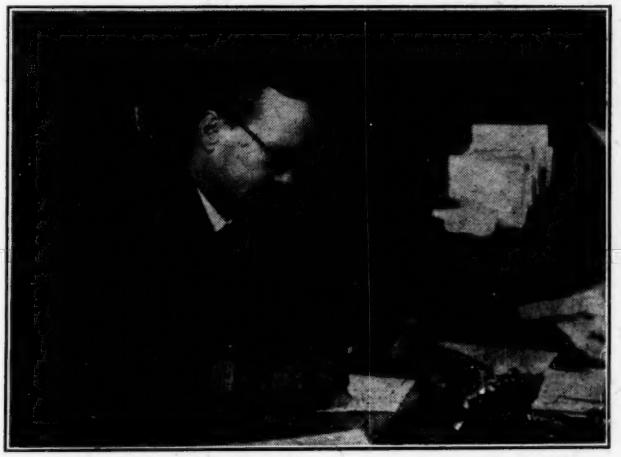
Treatment

This form of ringworm responds well to treatment. Whitfield's ointment may be rubbed into the affected patch twice daily for a fortnight or three weeks, and seldom fails. Recurrences are not very unusual and are probably most often due to a reservoir of infection on the feet or nails. In order to avoid reinfection from clothing, underwear should be boiled during the period of treatment.

Pityriasis Versicolor

It is difficult to say how common this type of ringworm is; very often it is found on patients who are being examined for another purpose, and, although some patients complain of slight irritation, many people do not notice its presence. The eruption appears principally on the back and front of the trunk and is made up of patches or sheets of a faint fawn or brownish colour. It is usually only when the patches are scraped that a fine adherent branny scaling becomes evident. The patches are well outlined, but there is no erythema or other alteration of the skin. When scales are examined microscopically, fine filaments of fungus can be seen together with clusters of spores.

From the absence of any inflammatory change it is clear that this infection is extremely superficial. Symptoms are negligible, and it is not easy to persuade patients to use messy ointments over a wide area of the body for such a trivial complaint, since there are indeed no complications. Because of this I usually give a lotion consisting of 50% spirit containing 1:2,000 mercury perchloride to be swabbed over the affected areas twice daily. Underclothes should be boiled during the period of treatment. In practice, the eruption is rather resistant to treatment, and shows a strong tendency to relapse; I suspect this is largely because it is so difficult to ensure that treatment is effectively carried out.



LORD HORDER 80

Lord Horder, who is 80 years old on January 7, can look back on a life of unrivalled service to the profession and the public. Still having the physical energy and mental alertness of a man many years younger than himself, he can, too, look forward to giving—and we to receiving—many years of active and pertinent counsel in all those various aspects of life and medicine which have engaged and held his unflagging interest. As was said of him in the *Sunday Times* a year ago, "The societies of which he is either president or chairman are mainly those which support the right of an individual to decide for himself those matters which concern only himself." Some men prominent in public life come to assume a public mask. When on the public stage they play a part which has been carefully studied and rehearsed. But Lord Horder, on public and private occasions, has always been himself, forthright and direct in speech that at times has the precise sharpness of the etcher's tool. He has been and is an individualist in the best meaning of this word. It is surely this, added to his brilliant qualities of mind and unusual capacity to observe what he sees, which has made him the supreme clinician of his day. It was an almost uncanny observation that led him to supply a critical piece of evidence when he was called in during one of the illnesses of Edward VII.

It is just over forty years ago that Dr. Thomas Horder (as he then was) published his classical description of subacute bacterial endocarditis. He was the first to isolate a streptococcus from the blood in this disease and establish it as an infective condition. This was the fruitful outcome of his early awareness of the importance of clinical pathology, the subject of his first published book in 1910. In the early years of his consulting practice he "did" his own pathology, with the help of what came to be known as "Horder's box," containing test-tubes, culture tubes, blood-counting apparatus, and a blood-culture syringe. He brought clinical pathology to the bedside at a time when too many were content to bring only the bedside manner. Lord Horder's clinical exemplar was that great Bart's physician and teacher to whom he was house-physician, Samuel Gee, and Bart's holds in grateful memory Gee's house-physician who in the course of time also became a great physician and teacher in an institution which for over 800 years has been an active witness to the continuity of medical history.

The International Hospital Federation's *News Bulletin* reports some differences from British practice noted in a tour of Swedish hospitals. Bedpans are never sterilized, a good dousing in cold water being considered sufficient. Creeping plants in pots and boxes are often trained up the inside walls of wards and hospital rooms; the patients like it, and doctors and nurses see no harm in it.

MEDICAL NEW YEAR HONOURS

The names of the following members of the medical profession were included in the New Year Honours List published in the *London Gazette* on January 1.

K.C.V.O.

DANIEL THOMAS DAVIES, C.V.O., M.D., F.R.C.P. Physician to the King.

FRANCIS HUGO TEALE, C.V.O., M.D., F.R.C.P. Honorary Officer in charge of Immunotherapy Department, University College Hospital, London.

K.B.E. (Civil Division)

PERCY SELWYN SELWYN-CLARKE, C.M.G., *M.C.*, M.D., F.R.C.P. Governor and Commander-in-Chief of the Seychelles.

Hon. K.B.E. (Civil Division)

FRANCIS AKANU IBIAM, Hon. O.B.E., M.B., Ch.B. Member of the Executive Council, Nigeria.

Knighthood

KOFOWOROLA ADEKUNLE ABAYOMI, M.D. Member of the Executive Council, Nigeria.

FRANK MACFARLANE BURNET, M.D., Sc.D., Ph.D., F.R.A.C.P., F.R.S. Director of Walter and Eliza Hall Institute, Melbourne, Australia. For services to biological research.

WILLIAM KELSEY FRY, C.B.E., *M.C.*, M.D.S., F.D.S., M.R.C.S., L.R.C.P. Dean of the Faculty of Dental Surgery of the Royal College of Surgeons of England.

GORDON MORGAN HOLMES, C.M.G., C.B.E., Hon. Sc.D., Hon. D.C.L., M.D., F.R.C.P., F.R.S. Consulting Physician, Charing Cross Hospital and National Hospital for Nervous Diseases, London.

C.B. (Military Division)

KENNETH ALEXANDER INGLEBY MACKENZIE, B.M., B.Ch. Surgeon Rear-Admiral. Honorary Physician to the King.

THOMAS YOUNG, O.B.E., M.B., Ch.B. Major-General, late R.A.M.C. Honorary Physician to the King.

C.M.G.

CLIFFORD CRAIG, M.D., M.S., F.R.A.C.S. Surgeon Superintendent, Launceston General Hospital, Tasmania.

C.V.O.

GEORGE PROCTOR MIDDLETON, M.V.O., M.B., Ch.B. Surgeon Apothecary to His Majesty's Household at Balmoral.

C.B.E. (Military Division)

VYVYAN STEWART EWING, M.B., Ch.B., D.P.H. Air Commodore, R.A.F.

C.B.E. (Civil Division)

JAMES BOYD, M.D., F.R.C.P.I., D.P.H. Chief Medical Officer, Ministry of Health and Local Government, Northern Ireland.

LUCY MEREDITH BRYCE, M.B., B.S., F.R.A.C.P. Honorary Director of the Blood Transfusion Service, Victoria, Australia.

JOSEPH STANLEY HOPWOOD, M.B., B.S. Medical Superintendent, Broadmoor Institution.

PETER JAMES KERLEY, M.D., F.R.C.P., F.F.R. Director, Radiological Department, Westminster Hospital. Consultant Adviser on Mass Radiography, Ministry of Health.

GEOFFREY MARSHALL, O.B.E., M.D., F.R.C.P. Principal Medical Referee to the Civil Service Commission.

KAMIL MOHAMED ARIFF bin KADIR MUSTAN, L.M.S. For public services in the Federation of Malaya.

ADAM RANKINE, *M.C.*, M.B., Ch.B. Lately Colonial Medical Service, Director of Medical Services, Mauritius.

VINCENT BRIAN WIGGLESWORTH, M.D., F.R.S. Director of the Unit of Insect Physiology, Agricultural Research Council.

O.B.E. (Military Division)

JOHN ALISTAIR MANIFOLD, M.R.C.S., L.R.C.P. Lieutenant-Colonel, R.A.M.C.

HENRY LINDSAY ROXBURGH, M.B., Ch.B. Acting Wing Commander, R.A.F.

O.B.E. (Civil Division)

JAMES FANSTONE, M.D. Physician, Surgeon and Founder of the Hospital Evangelico, Annapolis-em-Goyaz, Estado de Goyaz, Brazil.

THOMAS GILRAY, M.B., Ch.B. A medical practitioner of Napier, New Zealand.

DON SAM de SIMON, L.R.C.P.&S.Ed. Medical Superintendent, Leprosy Hospital, Hendala, Ceylon.

JOHN ALEXANDER JAMES, Ch.M., F.R.C.S. A prominent physician and surgeon in the Australian Capital Territory.

ALEXANDER CAMERON MCKILLOP, M.B., Ch.B., of Christchurch, New Zealand. For services to psychiatry.

WILLIAM HUGH CLIFFORD PATRICK, M.D. Regional Medical Officer, Ministry of Health.

MOHAMED ALI RANA, M.B.E., M.R.C.S., L.R.C.P. For public services in Kenya.

ANDREW JAMES SHINNIE, M.D., D.P.H. Medical Officer of Health, City of Westminster.

SINNATAMBY SUBRAMANIAM, L.R.C.P.&S.Ed. For medical and social services.

ALAN ROBERT STANLEY VICKERS, Ch.M. For services to the Flying Doctor Service in Australia.

LEON JAMES WILLIAMS, M.D. For public services in Bermuda.

M.B.E. (Military Division)

HAMILTON JAMES ELVERSON, B.M., B.Ch. Major (temporary), R.A.M.C.

WILLIAM BOYD JACK, B.M., B.Ch. Acting Interim Surgeon Lieutenant-Commander, R.N.

TERENCE RICHARD JANSEN, E.D., L.R.C.P.&S.Ed. Major, Ceylon Medical Corps.

TREVOR STANSFIELD LAW, M.B., Ch.B. Surgeon Lieutenant, R.N.

M.B.E. (Civil Division)

ARTHUR GRIFFITHS FARR, M.B., B.Chir. Colonial Medical Service. Acting Senior Medical Officer, Tanganyika.

SAMUEL FERGUSON FLOYD, M.B., B.Ch. General Medical Practitioner, County Down.

AGNES GRACE GILLESPIE, M.B., Ch.B. Medical Missionary, Mary Calvert Holdsworth Memorial Hospital, Mysore City, India.

JOHN JAMES SPARKS, L.M.S. British Vice-Consul at Coatzacoalcos, Mexico.

FRANCIS HENRY WHITEHEAD, M.R.C.S., L.R.C.P. Chairman, Wandsworth and Battersea War Pensions Committee.

At a pathological meeting of the Liverpool Medical Institution on Thursday, December 14, Dr. R. E. Rewell, pathologist to the London Zoo, read a paper on "The Pathology of Captive Wild Animals." He said that the size, shape, and metabolism of cells was limited and controlled by factors independent of the dimensions and structure of the whole animal. Thus a granulosa-celled tumour of the ovary had the same structure and might show the same variations in a small monkey as in a woman, or as in a gigantic whale. However, such tumours could be found only in animals with Graafian follicles. Variation in the structure of organs conditioned local variations in the results produced by disease. Inflammation of the lung of a reptile, which has no closed spaces, produced a condition comparable to enteritis in mammals; the lung of a bird, with its "through and through" arrangement, became completely solid; while the numerous blind ends of a mammalian lung gave rise to the characteristic lobular consolidation. Differences in metabolism must be responsible for such things as the high incidence of Laennec's cirrhosis of the liver in the Arabian camel, not seen in the closely related Bactrian camel, though both are usually on the same diet while in a zoo. Skin boils in ungulates were due to a corynebacterium, while in Equidae arterial aneurysms were caused by the worm *Strongylus vulgaris*. Some of these pathological conditions might be useful in the selection of suitable experimental animals outside the range of those commonly employed.

Reports of Societies

ROYAL SOCIETY OF MEDICINE

AETIOLOGY OF PNEUMONIA

The subject of pneumonia was discussed by the Section of Epidemiology and State Medicine of the Royal Society of Medicine on Friday, December 15, 1950.

Dr. W. H. BRADLEY, from the chair, said that the comparative mortality index of pneumonia (all forms), taking 1938 as 1.0, had been 1.76 in 1911–15 and was 0.5 in 1948. There had been some fall even before the introduction of the sulphonamides in 1935. Deaths from all forms of pneumonia were 26,786 in 1935 and 20,792 in 1949. Notifications were 46,401 in 1935 and 34,538 in 1949. Pneumonia remained a very serious problem.

Dr. J. G. SCADDING pointed out that it was impossible to classify pneumonias purely on an aetiological basis. Certain pneumonias were due primarily to a breakdown of the defences of the respiratory tract. When this happened organisms in the upper respiratory system not normally pathogenic might gain access to the alveoli and there cause an inflammatory reaction. He therefore divided pneumonias into two broad groups, the acute specific pneumonias and the aspiration pneumonias. In the former group were included pneumonias due to specific bacteria, viruses, rickettsiae, fungi, plasmodia, chemical substances, and allergy. He also included in this group pneumonias associated with viruses such as those of influenza and measles and due to the combined action of viruses and bacteria. Aspiration pneumonias were preceded by an upper respiratory tract infection or by bronchitis. Commonly the illness began insidiously and respiratory symptoms were prominent, in contrast to the typical virus pneumonia in which constitutional symptoms predominated; but it was admitted that in many cases the clinical differentiation was difficult.

Dr. Scadding gave an account of aetiological studies carried out by his group at the Postgraduate Medical School of London since October, 1949. There were 110 cases of pneumonia. He contrasted the low incidence of pneumococcal pneumonia (26%) in 1949–50 with the much higher incidence (68%) at the same hospital in 1938–9. Most pneumococcal pneumonias occurred during the winter months, but they continued sporadically during the summer. All 10 staphylococcal pneumonias were seen between October and April. There were eight cases with cold agglutinin titres of 1 : 256 or above, and these were all much younger than the average age for the series; there were no constant clinical features and the cases were distributed throughout the year. There were a few cases with serological evidence of ornithosis or Q fever. The influenza B cases occurred from January to April, 1950, and were associated with pneumococcal or staphylococcal pneumonias, or with those of undetermined aetiology. In no fewer than 27 cases, which had had no chemotherapy before admission, no aetiological agent could be identified. Eighteen were scattered lobular or segmental pneumonias, and most of the patients either had had a preceding upper respiratory tract infection or were chronic bronchitic subjects. It therefore seemed likely that some at least were essentially aspiration pneumonias.

Professor C. H. STUART-HARRIS considered that the majority of infections of the lung were due to bacteria, but he stressed the frequent association of pneumonia with upper respiratory tract infections at all ages. He had found a fairly close relationship between the pneumonia death rate from season to season and the deaths in the great towns recorded as due to influenza. With his colleagues he had studied cases of pneumonia and other respiratory diseases admitted to hospitals in Sheffield between October, 1947, and April, 1949. He contrasted two phases within this period. The first phase extended from October, 1947, to December, 1948, during which time only two sporadic infections with influenza A virus were recorded among some 85 cases studied. Seventy-seven of the cases studied had had pneumonia and there were three deaths. During the second phase, from January to April, 1949, there was an epidemic of influenza A. In this phase 95 cases were studied; there were 70 cases of pneumonia with 16 deaths. There was some contrast in bacteriology between the two phases; staphylococcal infections were much commoner in the second phase. Of nine cases from which influenza A virus was recovered at necropsy the lungs were infected with staphylococci in six. During this phase serological tests for influenza A were positive in 27 out of 57 cases of upper respiratory tract infection, in 11 of 53 cases of non-fatal pneumonia, and even in four out of seven cases of heart failure. Though some cases of staphylococcal and influenza A infection ran a fulminating course, suggesting a symbiotic attack by the two organisms, cases of combined infection with pneumococci and influenza A seemed no more ill than those infected with pneumococci alone.

Dr. B. P. MARMION discussed serological tests for influenza, cold agglutinins, streptococcus MG, Q fever, and the psittacosis–ornithosis groups. Sera were submitted to the Central Public Health Laboratory at Colindale from all cases of pneumonia admitted to two large hospitals and from various cases of respiratory infection elsewhere in the country. Between June, 1949, and May, 1950, 24% of sera from cases clinically diagnosed as "influenza" or "febrile catarrh" were positive for influenza B; 14% of cases of lobar pneumonia were positive and 16% of cases of "atypical pneumonia." Twenty-one per cent of cases diagnosed as "atypical pneumonia" had positive cold agglutinins and 5.6% were positive for Q fever. The 26 cases serologically diagnosed as ornithosis were evenly distributed between the different clinical diagnoses, suggesting a lack of any well-defined clinical picture. Of these cases less than half had any clear evidence of contact with pigeons, chickens, or parrots.

In this series cases positive to serological tests for ornithosis and Q fever were evenly distributed throughout the year. The influenza B cases occurred between January and April, 1950. Cases with cold agglutinins were seen at all seasons but were more frequent between December and April. Positive titres for streptococcus MG occurred only in those having significant titres of cold agglutinins.

Dr. Marmion discussed the significance of the test for cold agglutinins. This test was positive in a high proportion of volunteers infected by the American Commission for Acute Respiratory Infections with filtered material from "primary atypical pneumonia." It was clear, therefore, that the reaction was associated with a virus infection. Eaton has brought forward good evidence in favour of his cotton-rat virus being the infecting agent responsible, but the evidence could not yet be regarded as conclusive. The position of streptococcus MG was uncertain. It was not clear whether the organism caused pneumonia itself; or whether it was symbiotic with a virus, in the same manner as in swine influenza; or whether the streptococcus MG shared an antigen with the causative virus.

WHAT IS GENERAL PRACTICE?

The first regular meeting of the newly formed General Practice Section of the Royal Society of Medicine was held on December 20, 1950, under the chairmanship of Dr. G. F. ABERCROMBIE, when there was a discussion on "What is general practice?"

Dr. G. O. BARBER (Great Dunmow), honorary secretary of the Section, said that when the Section was being formed this question was asked again and again, so that it seemed logical at the first meeting to face it and to define medicine as practised by the family doctor in so far as it differed from hospital and specialist practice.

He recalled an incident of 20 years ago. He was a young doctor and the old practitioner was dying. While he was in a little room, combined waiting-room and dispensary, behind a partition of wood and frosted glass, he heard one woman who was waiting say to another, "I don't think

much of this young doctor. They say he *examines* people to find out what is wrong with them." It had taken him many years to realize that that good lady was right. Not that one should not examine one's patients and keep records of them, but what had been borne in upon him was that the good practitioner, in living constantly among his patients, sharing their life in village or town, knowing them and their work, the conditions under which they lived, their pursuits, vices, love affairs, and so on, got so intimate a knowledge of them that long and detailed examinations became superfluous in a number of cases.

The Jungle and the Zoo

The general practitioner, Dr. Barber went on, was a naturalist, pursuing his quarry and studying its habits in the jungle; the specialist observed only the specimens in the zoo. To put it in another way, the study of the one was *in vivo* and of the other *in vitro*. The essence of general practice was the study of man in his natural environment. The specialist was handicapped by having to take a one-sided answer to his questions, whereas the general practitioner knew many of the answers automatically. He knew his patient's heredity as well as his environment, and that was often helpful, for instance, when the stress syndrome began to develop. One observed different members of a family tending to break down under the same stress or under different stresses in the same way.

As medicine and surgery and the allied specialties became more complicated and their technique more and more a matter for experts who were at the same job all the time, many believed that the general practitioner was doomed to the role of an intelligent signpost. He believed directly the opposite. The patient could not be divided, and if any special aspect of him had to be investigated it was the duty of the general practitioner to oversee that investigation to the utmost limit of his understanding and to ensure that the specialty was applied to the patient in full knowledge of the patient's circumstances. The general practitioner must not send his patient to hospital and forget all about him until he came back again.

Responsibility in general practice was continuous, and sometimes took the practitioner a considerable distance away from general medicine. General practice involved the treatment not so much of the possible triviality which had caused anxiety in the home, and perhaps in the neighbourhood, as of the herd panic itself. It was for the practitioner to allay that anxiety as well as actually to diagnose and treat the case.

Birth and Death

At no time was this more marked than on the two great occasions of birth and death. At a birth there were more people in the house than the mother and the child. There were the expectant father and grandparents, and their treatment could be as great a trial to the general practitioner as the handling of the confinement itself. Death, again, was something which was the concern of general practice: there was not only the dying patient, who was usually the least conscious of the concern he aroused, but his family and friends. The way in which the death was handled by the general practitioner might make all the difference. In an age which must regretfully be described as irreligious, there often fell to the general practitioner on such occasions something of the function of the priest.

In chronic cases it was often said that nothing could be done. But general practice consisted in doing something to make life as bearable as possible for the patient, and to make him live as comfortably as he could with his disability. On the research side there were various matters which might engage the practitioner's attention, such as the observation of seasonal epidemics, not necessarily the serious epidemics, but the minor cases, the transient vertigo, nausea, and vomiting, the epidemic myalgia, the occurrence at certain times of a series of cases of acute backache or pain on turning the head.

Dr. Barber said in conclusion that general practice was still, and always would be, just what the individual general practitioner made it. It was not a question of definition by any medical body, but it was the general practitioner's own conception of service to his patients, exemplified by the standard of his work and his willingness to accept responsibility. It was the whole care of his patient in the normal environment and to the utmost extent.

Experts in Environment

Dr. J. D. SIMPSON (Cambridge) said that the general practitioner knew but little of elaborate techniques in diagnosis and treatment; he accepted them, and used them to advise or treat the patient in relation to his or her background, and it was of this environment that the general practitioner should have special knowledge.

Dr. Simpson gave instances in which the question of fitness for athletics came before the general practitioner, especially in a university town. On such occasions he must rely on his knowledge of the patient, the game itself, and his own immediate judgment.

Another problem was "fits and faints." Just as one swallow did not make a summer, so one transient loss of consciousness did not justify a diagnosis of epilepsy. In these days of the worship of scientific investigation practitioners were often asked to have tests carried out. An electroencephalogram would in these circumstances, in 9 cases out of 10, only confuse the issue, and so would the administration of phenobarbitone "to stop any further fits," unless the orginal attack had been seen by a really reliable witness and there was no question at all of the diagnosis. A quick reference to "fits" in a medical students' textbook would soon convince the patient that he had epilepsy with all its concomitant misery. How much better and more courageous to tell the patient that it was a "faint," and to wait, without giving drugs, until the next attack, which might never occur. Sometimes, of course, the doctor would be wrong, and there might even be tragedy, but he maintained that the number of patients saved from the fear of a disease which they had not got would amply justify this action.

Research in General Practice

Dr. Simpson added a word about research in general practice. The opportunities for research were twofold. There was first the long-term collection of cases of a particular disease. When planning a definite investigation of this kind the practitioner should seek statistical advice at the outset and use a special form for his notes so that they might be of real value. Nothing was more maddening than to look up a past case and find that the notes were incomplete, so that the case could not be included in a series.

His second opportunity for research was in the recognition of clinical syndromes when these occurred in epidemics. For example, the speaker had recently had a group of cases of gastro-enteritis with such violent intestinal colic that heavy sedation had been necessary. He had had 10 such cases within four days. Description of such outbreaks at meetings of the Section would be of the greatest value.

In conclusion he summarized his views of the special responsibilities of the general practitioner as follows: (1) he should be expert in the environment of his patients; (2) he should have the courage of his convictions and not be afraid of accepting responsibility when this would save his patients fear and anxiety; (3) he should bring to the new Section of General Practice notes of the pattern of common diseases so that general practitioners could learn by discussion in the same way as their specialist colleagues.

"Only a G.P."

Dr. E. P. SCOTT (Marylebone) declared that general practitioners were the backbone of the medical profession. There had been a slightly derogatory feeling about people who were in general practice—they were "only G.P.s"—but

this was unjustified. There were certain attributes which every practitioner who wanted to be successful must possess. The most important was personality; the second, a knowledge of his work. Perhaps the third should be luck, and the fourth a combination of a warm heart and warm hands. He quoted Lord Dawson of Penn as having said to him, "The treatment of patients is 50% the treatment of the relatives." On the question of research in general practice he mentioned the achievement of Sir James Mackenzie, who did all his research work in Birmingham as a general practitioner.

Dr. C. H. BILLINGTON (Haileybury) spoke of the interest of general practice at a public school; it was the more stimulating because it was concerned with health rather than with illness. Dr. RAYNER (London) said that general practitioners very often did not pass on sufficient information when their patients went to the hospital or the specialist. Dr. LAWRENCE DULAKE (Reigate) said that the work of a general practitioner depended so much on the neighbourhood in which he lived, the kind of practice he followed, and his individual personality that generalization was difficult. Patients often expressed a desire to see a "proper doctor." The general practitioner was a "proper doctor." All the rest were incomplete doctors. Doctors had sometimes been divided into two categories—the "tut-tutters" and the "pooh-poohers." He specially deprecated the "tut-tutters," for they somehow communicated their own real anxiety to the patient. At least half of the patients coming to his surgery suffered from some psychiatric upset. Sometimes this was an overlay, but very often it was the main trouble.

The Bottle of Medicine

Dr. E. T. HUGHES (West Wales) said that when he looked at Sir Luke Fildes's picture "The Doctor" (the original was in the Tate Gallery) it made him feel very humble. There was much that they, the younger men, could learn from the old family practitioner. He himself succeeded a dear old country doctor of 83, and he learned a lesson a very few days after entering the practice. A woman came in for a bottle of medicine, and with some pride he dispensed it quickly and deftly from the bottles on the shelf. The woman looked at it with suspicion. She evidently thought he had left something out. Later on he had an opportunity of watching how his old principal did it. He fingered the bottles carefully, choosing one and then another with great deliberation, and the pipe in his mouth added a certain leisurely dignity to the proceedings. Probably the impression it made on the patient was more valuable than the ingredients themselves. General practice, said Dr. Hughes, was the practice of doctoring. The specialist went into medicine as an intellectual pursuit, and thereby lost some of the sense of vocation.

Telling the Patient

Dr. L. W. BATTEN (Hampstead) said that general practitioners were the only people in the profession who looked after the whole person, and he did not think this could be done by teams. One of their jobs was to try to explain to the patient what was the matter with him, a thing which he was not told in hospital. A cause of the frequent dissatisfaction with hospital treatment was that, although the patient received proper attention, he often came out without ever learning the nature of his illness, and accordingly felt that he had been deprived of something. People sometimes spoke as if it was easy to persuade patients to be well. General practitioners knew how difficult it was. It was as big a sin to start an imaginary disease as it was to overlook the beginnings of a serious one. The general practitioner had not only to introduce the specialist to his patient, but in a sense to protect the patient from the specialist. Sad would be the lot of the patient in the hands of the specialist without the general practitioner in the background.

Dr. M. C. ANDREWS (Wembley) said that it was not quite fair to suggest that the consultant treated the patient in the "zoo" whereas the general practitioner treated him in the "field." A great deal could be done by getting the consultant to come as often as possible to the houses of patients, and for that reason he tried to limit the number of consultants he called in, and thus they got to know, not merely the patients, but himself and his own personal attitude to his patients. Dr. Andrews thought that broadcast features such as the "Radio Doctor," and also articles by "Nurse So-and-So" in women's journals, could well be dispensed with, but that much good might be done by bringing together groups of people who were suffering from the same disability, as was already done in some cases with the deaf or the partially blind.

Dr. E. M. DIMOCK (Broxbourne) said that general practitioners by the way they handled the family prevented a great deal of ill-health. He considered that the psychiatrists were making heavy weather with lots of incurables who might have been picked out and dealt with if general practitioners had had better encouragement from the medical schools. Dr. A. TALBOT ROGERS (Bromley) spoke of the general practitioner in committee work and in planning the medical service locally and centrally. Dr. St. G. B. DELISLE GRAY (Walthamstow) said that one of the troubles with general practice was that the young men in hospital did not know what general practice was. The important thing was that young men, before they specialized, should learn something about what general practitioners did in the course of their work. All men before they went into a specialty should spend 6 to 12 months in general practice.

Dr. BARBER, in replying on the discussion, said that of course he had not suggested that the general practitioner should refrain from examining his patient, but without the case history the examination might be futile. He agreed with Dr. Hughes about the value of the bottle of medicine. It should be borne in mind that the practitioner when he came into a home in which there was sickness brought a therapeutic influence, or should do so, but there was likely to be a return of anxiety after he had left. The bottle of medicine served as an emblem of his continuing authority.

The annual report of the Government Chemist (H.M.S.O., 1950, price 1s.) contains many matters of medical interest. The department acts as a kind of national public analyst, and has to solve such problems as how to detect horsemeat in sausages or wax in sweets, and to watch over the quality of national flour and dried milk, which it reports to be maintaining a high level. Indeed, the milling of the flour has been slightly improved, since there is a drop in its fibre content; the vitamin B_1 continues at just under 3μg./g., and 14 oz. chalk is added per sack. But research is also carried out. During the Olympic Games, 1948, diet analyses of the food eaten by 28 competitors of eight nationalities showed that the daily calorie intake of the athletes was on the whole lower than that found at the last Olympiad in Berlin. One Mexican was taking only 2,100 calories, while one British competitor had over 4,600. A suggestion that old worked-out gravel pits should be filled up with dust sieved from household refuse was studied to see whether land could thus be reclaimed, but analysis showed the dust to be up to 53% organic matter and experiment yielded a polluted water. Some of the investigations are more adventurous. Ambergris, the calculus found in the gastro-intestinal tract of sperm whales which have been feeding on cuttlefish, is being studied on behalf of the Discovery Committee. By the analysis of fluorine in fossil bones it is possible to go some way towards dating the skeletal remains of prehistoric man, and the department has been busy in this way on the Swanscombe skull and the remains of Piltdown man. An attempt to decide whether pottery found on an ancient British homestead about 2,000 years old was made from local clay was inconclusive; the *x*-ray diffraction patterns of clays and pots were studied, and trace elements estimated, in order to make comparisons of the composition of both pottery and local clay.

Correspondence

Appendicitis

SIR,—I read the article by Sir Cecil Wakeley and Mr. Peter Childs (December 16, p. 1347) with much interest, but with all respect I feel I must comment on one or two points, first out of fairness to my late chief, James Sherren, and secondly as a result of my own experience.

It is correctly stated that the number of deaths from acute appendicitis rapidly fell from 3,027 in 1938 to 1,970 in 1941. This was followed by a stationary period for three years. In 1945 a further fall began which is continuing, and the latest available figure (1947) shows 1,491 deaths. They also state that the two periods of improvement were due to the introduction of sulphonamides and penicillin respectively. If so, why the three-year mid-war period when there was no lack of supplies of antibiotics? A more likely explanation is that evacuated children were scattered over the country, commonly away from parental supervision, under the care of overworked and often elderly practitioners, and with only cottage hospital facilities in many cases. Sulphonamides and penicillin have doubtless helped, but other factors are of importance in this welcome reduction in mortality, as I have already suggested in a recent article (*Clin. J.*, 1950, 79, 182).

"Parents, practitioners, and surgeons all played a part in achieving this welcome improvement—parents because they now but rarely pour castor oil into unwilling stomachs, practitioners on account of earlier diagnosis and a greater sense of urgency, and surgeons because the danger of the rule-of-thumb method of immediate operation on all cases is increasingly appreciated, and expectant treatment is rightly considered in cases who are 'too late for the early operation, and too early for the late one.' Sulphonamides and penicillin also deserve credit in helping to control infection."

It is stated, "If the diagnosis is appendicitis, and if it is clearly not chronic, operation is urgent. There is no place for expectant treatment." Also we are reminded that, since I advocated expectant treatment in an article in 1937, "the following have been added to the surgeon's armamentarium: sulphonamides, penicillin, intravenous drip therapy, and gastric suction." It is obvious that these new additions are just as valuable in cases treated expectantly as for those operated on at any time. The three cases, referred to in the article, in which the expectant treatment of "appendix mass" failed were certainly unfortunate, but over a large series of cases at least 90% were found to subside with the recent additions to the surgeon's armamentarium.

As admitted in the article, appendicectomy is now always possible if an immediate operation is performed. It is stated, "Where an abscess was found surrounding the appendix the appendix was removed whenever possible"; also, in the case of an appendix abscess "the appendix was not removed unless it was readily accessible." These patients, having survived the exploratory operation, presumably submitted to a second operation for appendicectomy.

There are many other points one would like to raise, but space forbids, so I conclude with and endorse the statement of James Sherren, who after twenty years' experience at the London Hospital wrote in one of his last papers (*British Medical Journal*, 1925, 1, 727). "The only change I have made has been to greater conservation and more patience in dealing with cases of appendix abscess."—I am, etc.,

London, W.1. R. J. MCNEILL LOVE.

SIR,—Sir Cecil Wakeley and Mr. P. Childs are to be congratulated on their 217 cases of appendicitis without a death (December 16, p. 1347). A benign gremlin must be looking after their cases, for in combining a recommendation of the muscle-split incision with a condemnation of expectant treatment in late cases they are surely doing a disservice to surgery.

Admittedly the most important adverse factor in morbidity and mortality in acute appendicitis is late diagnosis. The next most important adverse factor is the indiscriminate employment of the muscle-split incision. This incision has been taught in our medical schools and practised all over the country since operation first became recognized as the correct treatment for acute appendicitis. Yet it is a bad incision, with little to recommend it.

The commonest cause of death in acute appendicitis is intestinal obstruction. This is due to peritonitis, either localized or generalized, and now happily amenable to considerable control by chemotherapy, gastric suction, and intravenous drip therapy. But the tendency to intestinal obstruction is aggravated by trauma to the oedematous tissues in the ileo-caecal angle at the time of operation, especially to the last inch of the ileum. In the presence of acutely swollen and oedematous tissues it is often very difficult to deliver the appendix from the retro-caecal or pelvic positions (the commonest) through the limited exposure of the muscle-split incision, and even if the incision is enlarged by the Rutherford Morison muscle-cutting method the decision to do so is made only after considerable trauma has occurred. The more skilful the surgeon the less is the trauma, but no surgeon of experience can look back over his career and not recollect many difficult struggles to deliver an appendix through a muscle-split incision. Moreover, the operation for acute appendicitis is *par excellence* the training operation for house-surgeons and registrars all over the country, and so long as they are taught to make this incision they too will struggle and learn by bitter experience, and the mortality rate from acute appendicitis will not improve.

It is not sufficient to make a diagnosis of acute appendicitis. The surgeon should assess in his mind as far as possible the position of the appendix and the probable extent of oedema and induration of surrounding tissues, the length of history being the most important factor to bear in mind. The incision should be planned accordingly. In 90% of cases it will be found that the right paramedian incision, extending from the level of the umbilicus to one inch above the pubis, will give an easy and adequate exposure through which the caecum and terminal ileum can be gently elevated and drawn towards the midline with a minimum of trauma, and the appendix removed no matter in what position it may be. Gentleness in the handling of acutely inflamed tissues is the key to the further reduction in mortality in acute appendicitis. It is also the key to the reduction of wound infection, which is more common in muscle-split incisions, which require retraction, sometimes forceful, than in paramedian incisions, which require none. If a localized appendix abscess in the right iliac fossa requires drainage, a muscle-split incision over it is the best approach. Similarly, in a late case in which the appendix is judged to lie in the high retrocaecal or paracolic positions, a Rutherford Morison incision *ab initio* is the best approach. In all other instances the paramedian incision is the best, particularly so for the pelvic appendix, in which correct diagnosis is so often delayed.

The muscle-split incision as a routine must go. It is a relic of the past, of the bad old days of indifferent anaesthesia.

The recommendation of the muscle-split incision for acute appendicitis is debatable. Its recommendation for chronic appendicitis is bad surgical teaching. The authors admit in Table IV that in 12 out of 39 cases so treated the appendices were normal on pathological examination. Why, then, the muscle-split incision, and what was wrong with these 12 patients? In *all* cases of co-called chronic appendicitis the paramedian incision should be made, so that the pelvic organs and terminal ileum and mesentery can be examined and the gall-bladder and kidney palpated. Many patients who have subsequent operations for pain in the right side of the abdomen already bear the tell-tale small scar in the right iliac fossa of which they and their surgeons were once so proud but with which they are now so disappointed.—I am, etc.,

Amersham, Bucks. ROBERT STRANG.

SIR,—Sir Cecil Wakeley and Mr. Peter Childs (December 16, p. 1347) have again focused attention on this important subject. I do not think it is sufficiently realized how ignorant the general public is of the symptoms of appendicitis, and how prevalent is the pernicious custom of taking a purgative, particularly castor oil, when abdominal pain comes on. Four recent cases have shown me how even

among intelligent well-educated people this dangerous trust in the castor-oil bottle persists, sometimes with disastrous results.

Case 1.—A woman, 75 years of age, had a central abdominal pain, accompanied by vomiting. The pain moved to the right iliac fossa next day. Her daughter, a school teacher, gave her a large dose of castor oil on the following day, and the symptoms became much more severe; her doctor, however, was not called in till the day after. A few hours later I removed her gangrenous perforated retrocaecal appendix; a faecolith was lying near by in the poorly walled-off abscess cavity in the right iliac fossa, and peritonitis was spreading. Drainage of the abdomen was necessary. Severe paralytic ileus ensued, and her condition was precarious in the extreme for a fortnight. Fortunately, with chemotherapy, gastric suction, a blood transfusion, and intravenous fluids, she rallied, and eventually made an excellent recovery.

Case 2.—A man aged 48 had "crampy pains" in the upper abdomen following a hearty dinner. Next day the pain had shifted to the right side. He took a large dose of castor oil, and, though his bowels moved, the pain grew steadily worse. He called in his doctor on the afternoon of the day following. By this time he was feeling pretty ill and was extremely tender in the right iliac fossa. At operation that evening I removed a perforated retrocaecal appendix from a well-walled-off abscess in the right iliac fossa, and drained the abdomen with a strip of corrugated rubber down to the appendix site. He made an uninterrupted recovery, after a full course of penicillin and sulphonamides.

Case 3.—A woman, aged 72 and extremely obese, had central abdominal pain and vomiting. Next day she took a large dose of castor oil. The pain became worse, and she was admitted to hospital late that evening. Her urine was loaded with pus cells, and she was observed for some hours as a possible case of right pyelitis. Tenderness and resistance increased in the right iliac fossa, and I operated on her on the following day. At operation a gangrenous perforated retrocaecal appendix was removed, and the abdomen and abdominal wall drained. She made an uninterrupted recovery.

Case 4.—A man aged 69 had central abdominal pain at 3 a.m. following a dinner party. He vomited, and in the morning he took a large dose of castor oil. The pain moved to the right iliac fossa, and became severe. His doctor was called in that evening at 7 p.m. Two hours later I removed an appendix lying in the subcaecal position, 6 in. (15 cm.) long, black the whole way, with a faecolith blocking it at the base, and the lumen full of dark fluid. Local peritoneal reaction was slight. Fortunately the removal of the appendix was easy, and recovery was uneventful.

These cases illustrate what has been noted frequently before; that castor oil, taken for abdominal pain which happens to be the start of an acute obstructive appendicitis, acts in the abdomen like a match applied to a powder magazine. It stimulates a vigorous peristalsis and so accelerates the progress of necrosis of the appendix wall at the site of the obstruction (usually a faecolith), with consequent perforation and discharge of the highly infected contents into a peritoneal cavity which sometimes has not had time to prepare effective barriers.

As no one can tell if colicky central abdominal pain is not the commencement of an attack of acute appendicitis, the administration of a purgative, particularly castor oil, in such a complaint is obviously an extremely risky and unwise procedure. There would seem to be considerable scope here, therefore, for members of the medical profession to enlighten and educate their patients, and so help to lessen the dangers of this disease, and also give the surgeon a chance to remove the appendix before it has perforated.—I am, etc.,

Edinburgh. J. A. ROSS.

Economics and Medicine

SIR,—In the many discussions that preceded the introduction of the National Health Service, speakers rarely talked finance, and when they did were not infrequently rebuked that we were rightly more deeply concerned with ethics and high principles. Some of us felt that in the course of time economics would enter the picture. Hopes were pitched high on the prospect of bigger and better hospitals, fine equipment, great opportunities, and no financial restrictions. All this might have been possible in an expanding and flourishing national economy. However, for many reasons, including inflation and the cost of defensive rearmament, the funds have not been available. Boards of governors have been placed on a restrictive budget, new buildings and equipment have been deferred, and expensive preparations such as penicillin and hormones curtailed to essential needs. As to general practitioners, financial terms of service have produced hardships, and serious negotiations are afoot. If inflation proceeds, these hardships will increase. Even a socialist medical association has sent a telegram to the Minister: "We want increased salaries and better working conditions." As to consultants, those who have looked entirely or largely to a fixed salary, by the week or by session, do not find financial conditions easy. Those who still look largely to private practice may have noted that "Harley Street," as always, tends to reflect general economic conditions and that higher fees proportional to inflation are not popular. Consultant trainees are finding few staff vacancies, as the hospital service has not been able to expand as was initially hoped.

What does the future hold? Current articles in economic and financial journals take the view that adequate increased taxation to meet the Government's financial burden is not feasible and is apt to defeat its own purpose. They turn to the question of cutting Government expenditure (although it is more realistically a problem of stopping it automatically rising), and they end: "How in these circumstances can the Welfare State survive?" Some point more directly to the National Health Service. No one knows the future, but all the trends point to the danger of a lower economic status for the doctor unless some reorientation is possible. I would suggest the need for a permanent economic advisory committee, including economists, to study these problems.

My own reflections are as follows: financial conditions for hospital residents and registrars were determined by the existence of a large body of ex-Service men. They should now revert, after an appropriate interval of notice, to those of the order believed adequate in pre-war days. Marriage for the young doctor could reasonably be deferred until the age of 30 unless special circumstances determine otherwise. Moderately senior consultants might consider whether they could give up some of their hospital sessions to make room for a proportion of the trainee consultants. This might involve some temporary hardship, but a return to a more balanced proportion of private work would in the long period prove generally beneficial and prudent.

It may be that sufficient private work would not come to all, and some reduction of fees instead of augmentation might call temporarily for harder work and less reward. In my view, under these circumstances there will always be a number of patients who are willing to pay for such private consultations, and the argument that there are not enough consultants for the national health scheme no longer holds. This might also be the trend of events in general practice. Interest in insurance and provident schemes for meeting illness should be revived and encouraged. The pendulum has swung too far and too fast, as the late Lord Dawson feared it might; and its correction will relieve the burden of the Government and the hospitals, and leave the profession in a more prudent position to maintain their proper economic and social status, and to meet the adversities of economies as well as relentless economics.—I am, etc.,

London, W.1. S. LEONARD SIMPSON.

A Co-ordinated Policy

SIR,—The outcry which has been aroused by the reduction of the registrar establishment seems to me to show up the inconsistencies of our own attitude to medicine to-day and to the National Health Service. On the one hand we cry out that over-specialization is diminishing the scope and lowering the status of the general practitioner, and on the other that more and more specialists are required. We say that the general practitioner should have fewer patients and a higher capitation rate, which means more money, as also does increasing the number of specialists. Is it really likely

that the country can afford any larger sum for the Health Service? Is it not more likely that any increase of the allotment for the above purposes would have to come by readjustment of the present rate of remuneration? Ought we not to have a co-ordinated policy, with priorities?

We deplore the gap between general practice on the one hand and hospital and specialist practice on the other, but any attempt at, or suggestion for, bridging the gap is effectively opposed not by the administrative side of the Service but by one side or other of the profession.

It is said, too, that the hospitals cannot function without their present number of registrars. But do they function any more efficiently than they used to do with smaller numbers? In spite of a medical and clerical staff greater than ever before it would appear to be usual for at least a week to elapse after the discharge of a patient from hospital before a letter is sent to his or her doctor. Investigations are necessarily more searching than they used to be, but much time and money are being wasted by blunderbuss investigations and by patients proceeding from one specialist to another. (Is it necessary for a physician to refer an asthmatic to an allergist for skin tests?) There seems to be a tendency for consultants to hold appointments at as many hospitals as possible, with the natural result that they are unable adequately to supervise the work at any of them. How rare, too, it would appear to be in some clinics for an out-patient to get beyond the registrar, and yet it is often for the consultant's experience and opinion that the patient is sent by the general practitioner. Is not the term specialist becoming more appropriate than that of consultant?—I am, etc.,

K. C.

B.C.G. and Sarcoidosis

SIR,—I was interested in Dr. F. E. Crawley's report (December 16, p. 1362) of three cases of erythema nodosum as a manifestation of Boeck's sarcoidosis.

The recent availability of B.C.G. gives the physician additional help in proving a diagnosis of sarcoidosis. Sulzberger and his co-workers (*J. invest. Derm.*, 1949, **13**, 249) have shown the differences in local response to B.C.G. in cases of congenital and acquired tuberculin anergy. Secondly, B.C.G. vaccination will not convert the positive anergy of sarcoidosis to normal tuberculin sensitivity (normergy).—I am, etc.,

Edgware, Middlesex. C. D. CALNAN.

Erythema Nodosum

SIR,—Dr. F. E. Crawley's interesting paper (December 16, p. 1362) once more raises the question of the aetiology of this disease. Perhaps my experience may throw some light on this. Since 1945 I have been treating erythema nodosum with vitamin K, with the result that the condition has cleared up in from one to three days. The dose of the vitamin appears to have little influence: 10 mg. of the synthetic vitamin twice daily appears to be as effective as larger doses. Those cases seen in the first few days cleared up within 24 hours. Those who had been ill for weeks or months took up to three days. Unfortunately I have only had eleven of these cases in five years, and up to a few months ago was not in a position to obtain accurate prothrombin estimations.—I am, etc.,

Belfast. T. A. KEAN.

Waiting-lists for Tonsillectomy

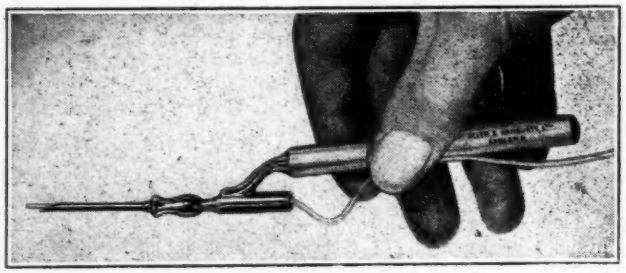
SIR,—It is a matter of concern how the many children waiting to have their tonsils and adenoids removed are to be treated in view of the lack of hospital beds in which to put them. In many localities the waiting-list runs into thousands, and children have been waiting so many years that we may expect to see their offspring appearing in the E.N.T. clinics soon. Some I have seen grow out of their disability, and doubtless many children could be better treated by convalescent treatment in the country with proper diet and environment, but this seems to be unobtainable. New Zealand, I read, has arranged these facilities, and I think if we had them we could save a lot of hospitalization, with its greater cost. I would like to put in a plea for a consideration and development of this type of treatment for patients *before* they become in-patients.—I am, etc.,

London, W.1. C. HAMBLEN-THOMAS.

Introducer in Blood Transfusion

SIR,—Since we have used polythene tubing for blood transfusion or other intravenous work it has been found that the introduction of a needle into a vein and threading polythene tubing through it is a thoroughly clumsy procedure. I have therefore designed an introducer, which is illustrated here.

The handle of the introducer is about the size of a 5-ml. hypodermic syringe and has the same feeling when handled. It gives complete control of the needle. The male end of the introducer is a Record size to fit the ordinary



M.R.C. donor needle, through which either size 1 or 2 polythene tubing can be inserted. The handle of the introducer is grooved (which is not seen in the photograph), and the tubing lies in it. When the handle is gripped the tubing is therefore prevented from slipping backwards and forwards in the needle. When the needle has been inserted into the vein the introducer is disconnected, and the polythene tubing introduced as far as necessary through the needle. The needle is then removed, leaving the polythene tubing in the vein, and is taken off the other end of the tubing so that the tubing can then be connected to the giving apparatus. This polythene introducer was made for me by Allen and Hanburys, and has been thoroughly tested and found to be extremely satisfactory.—I am, etc.,

ALEC E. BLAXILL,
Blood Transfusion Officer.

Colchester.

Death from Exsanguino-transfusion

SIR,—From a study of the literature of exsanguino-transfusion in adults, and from correspondence with its chief exponent, Dr. M. Bessis, it would appear that the manœuvre is almost entirely free from risk. We consider, therefore, that a fatality arising from this operation should be reported.

In a woman of 44 suffering from acute monocytic leukaemia an exsanguino-transfusion calculated to replace 95% of the patient's blood was planned. Careful physical and radiological examination prior to the operation failed to reveal any concurrent disease.

Thirty-five pints (19.6 litres) of blood were exchanged at a single session over the course of six hours, the patient being heparinized and the blood given into both arms, while withdrawal was effected by a wide-bore polythene cannula in the left femoral vein. Prior to the operation the patient had received six pints (3.4 litres) of packed cells, which had raised her haemoglobin to 13 g. %. During the course of the transfusion the patient developed symptoms of tetany, which were promptly relieved by intravenous injections of calcium gluconate. These symptoms recurred on three occasions, and each time required a larger dose of calcium gluconate intravenously to effect relief. At the end of the exsanguino-transfusion symptoms of tetany again became manifest, and, although they slowly subsided after administering a total of 120 ml. 10% calcium gluconate, the patient died.

Post-mortem examination confirmed the diagnosis of acute monocytic leukaemia, but also revealed tuberculous infiltration at the apex of the right lung, with very early miliary tubercles in the kidneys and spleen. No other notable changes were found.

We believe that this patient died a "biochemical" death due to the transfusion, and that tuberculosis played little or no part in the outcome. The biochemical changes resulting from perfusions of large quantities of citrated blood have not been adequately worked out. Certainly the amount of calcium required may be very much greater than the literature would suggest; and until more accurate knowledge is available we feel that a warning should be given of the possible dangers of exsanguino-transfusion in adults.

We are indebted to Drs. A. H. T. Robb-Smith, H. B. M. Lewis, and V. T. Pearce for their willing co-operation.

—We are, etc.,

P. D. BEDFORD.
L. G. LAITHA.

Oxford.

Salicylates in Post-tonsillectomy Analgesia

SIR,—In view of the fact that salicylic compounds reduce the blood prothrombin level. S. L. Fox and G. B. West (*Ann. Otol., etc., St. Louis*, 1948, **57**, 1032) state that the use of acetylsalicylic acid is a major factor in secondary tonsillar bleeding following tonsillectomy. Furthermore, the haemorrhagic complications following the administration of salicylates may involve the escape of hypoprothrombinaemic blood through capillary walls damaged by C-hypovitaminosis, since salicylic compounds combine with and hasten the excretion of vitamin C.

Earlier this year I had my tonsils dissected, and the resulting severe pain and discomfort were rapidly dispelled by sucking tablets containing acetylsalicylic acid, "menaphthone," and ascorbic acid ("ekammon"), which were recommended by the E.N.T. specialist, and no haemorrhagic complications developed. S. Shapiro (*J. Amer. med. Ass.*, 1944, **125**, 546) estimated that 1 mg. menaphthone will counteract the prothrombin-reducing action of 1 mg. acetylsalicylic acid, and therefore these tablets can be employed, even in massive doses if required, without the risk of inducing hypothrombinaemia and C-hypovitaminosis.

I am relating my personal experiences with the sole intention that others may be able to benefit from the analgesia afforded by salicylic compounds and at the same time be free from secondary haemorrhagic complications.—I am, etc.,

H. LEAHY.

Haslingden, Lancs.

Tuberculosis and the Health Service

SIR,—Professor F. R. G. Heaf indicates (December 16, p. 1353) an approach to the problem of tuberculosis of great value, but since his methods require extra clinics, extra medical staff, lay staff, and ambulances they will find little application at this present time. However, I think there is a solution which is not only more economical, but one which will improve the whole standard of medical care beyond the local problem of tuberculosis. It is for the general practitioner to practise more medicine.

The specialists in all branches, looking at the small field of their own specialty, continue to press for more buildings, more clerks, more equipment, and a great deal more money, as hospital finances show. And still they proclaim that the general practitioner is "the backbone of the profession." The general practitioner alone can integrate this health service, which the clinical specialties, the local health authority, the county health authority, and all the other highways and byways of medicine are disrupting through their specialism.

The home treatment of tuberculosis, indeed the home treatment of any disease, is the job of the general practitioner and the district nurse, the two most economical units of the Health Service. My principal and I have already three cases under treatment with *para*-aminosalicylic acid and dihydrostreptomycin, in consultation with the chest clinic. One, as Professor Heaf points out, has probably avoided sanatorium treatment completely, and has had her disease cut short by months. With more time the general practitioner could do far more artificial pneumothorax (A.P.) refills, for example. Now this could be applied to many other aspects of medicine. All antenatal work should be done by the general practitioner and the abnormal case sent for consultation with the consultant obstetrician; all child-health work is in the general practitioner's province, and the difficult case can be referred to the paediatric consultant; similarly with varicose veins, asthma, the rheumatic diseases, venereal diseases, etc. There is no need for a separate doctor, separate building, hordes of nurses, typists, and clerks. This could lead to general practitioners fully occupied in medicine and true consultants, rather than to the half-world of small-time specialists.

In this instance of tuberculosis the chest physician should be a consultant, preferably of all diseases of the respiratory tract, with hospital beds under his care. He sees the patient, advises on treatment (which the general practitioner carries out at home), and admits the patient for short periods for special treatment, induction of A.P., phrenic crush, etc. No need for ambulances—the general practitioner already has them available; no need for the "staff of the chest clinic to pay frequent visits"—the general practitioner does that too. Health visitors I am very sceptical about. A general practitioner with time to spare can give much better-informed advice in this highly technical phase of medicine.

My solutions, then, are:

1. A much-reduced maximum list carrying a similar income to the present. This, of course, will mean more general practitioners.
2. A reduction in the public health and special clinic services. These are responsible for much duplication we can ill afford.
3. A discouragement of some of the nonsense in general practice. A scheme similar to the Australian, where a list of about 170 urgent and effective drugs are prescribed free, and the rest are paid for.
4. This is not essential, but just a personal view: a salaried service. Away with the cannibalism. I think this would make general practice much happier, and would help one to treat patients as one thinks they should be treated, and not with an eye to gaining or losing "heads."

—I am, etc.,

P. M. VASEY.

Pontypridd.

SIR,—All members of the B.M.A. must welcome the publication of the Tuberculosis and Diseases of the Chest Committee's report (December 16, p. 1382). It is a good report, but it does not go far enough in my opinion on the side of prevention, although Professor F. R. G. Heaf's and Dr. G. F. B. Page's articles (December 16, pp. 1353 and 1380) help to fill the gap.

The scheme under the N.H.S. is uncoordinated and unbalanced: it is not that there is too much treatment; there is too little prevention. The link between the two is all but severed. It is the duty of the Ministry of Health to produce a co-ordinated scheme; this it is not doing. As I stated at the B.M.A. deferred Annual Meeting, the attempt is being made to fight tuberculosis on the cheap, and, while this is so, little progress can be made. Payments, urgently needed, have to be fought for in regional boards and management committees against all other interests, from tonsils and adenoids to geriatrics, and in the final allocation between treatment and prevention the claims of the latter may easily be overlooked. If a special tax of 10% or so were placed on the sale of television sets, a sheer luxury, and definitely allocated to tuberculosis services, it would go far towards providing the needed funds.

The preventive or epidemiological aspect should be given the first consideration; it pays double dividends. If smallpox or diphtheria had been regarded in the fatalistic way that tuberculosis is we should never have vanquished them, and, *mutatis mutandis*, we shall only cut the incidence of tuberculosis by one-tenth if we attack it epidemiologically by locating the source, and preventing the spread, of infection, and by increasing specific resistance. Every new case has contacts, who should at least be tested radiologically and by tuberculin, and if negative offered B.C.G. A child with a primary infection is often such a case.

It is true that we do not know the exact value or duration of the increased resistance conferred by B.C.G. inoculation, but is that a reason for denying to contacts the protection, such as it is, that such inoculation indubitably affords? The fatality rate of tuberculosis in the first year of life is very high, 36% according to Wallgren, and falls rapidly till puberty, when there is again a sharp rise. It has been shown

in Denmark, particularly Copenhagen, that by vaccination of infant contacts with B.C.G. tuberculous meningitis in infants, one of the principal causes of infantile deaths, can be virtually stamped out. As Professor Gaisford once said to me of gastro-enteritis, "The children die and nobody cares."

It is not only the 400 weekly deaths—though the Home Office would not pat itself on the back if the police had reduced the weekly number of murders from 400 to, say, 390—there is also the immense loss of health and of working hours involved. Leaving humanity aside, prevention of tuberculosis on an adequate scale would be well worth while. The process of contact hunting and inoculation is time-consuming and requires enthusiasm. It is not being adequately done and cannot be done with existing staffs, medical and other; these should be trebled to reach the Danish standard. Sir Robert Philip, as Dr. Page points out, taught that tuberculosis was preventable and that the dispensary was the centre of any integrated system of treatment and prevention. The present N.H.S. scheme is quite inadequate on the preventive side. With an adequate preventive system fewer beds would be needed. Is it merely insular prejudice that prevents us from using B.C.G. adequately?

The resolution of the B.M.A. Annual Representative Meeting last September condemning the existing arrangements for tuberculosis was passed unanimously. At the summer scientific meeting Professor Heaf, perhaps our highest authority, stated that we must "press for an amending Act . . . that would enable a service to be developed that could reduce the morbidity and mortality of this disease to an insignificant figure. Until this is done we shall proceed in a wasteful, discontented, and disorderly manner without hope of any real progress." As the Scottish Committee's report said, this crisis must be met in the spirit of a crusade. What can we do to rouse the Ministry out of its complacency to tackle this national scandal.—I am, etc.,

Heswall, Cheshire. C. O. STALLYBRASS.

Heart Cases in Mass Radiography

SIR,—Dr. Peter O. Leggat's article (December 16, p. 1364) raises points of considerable interest to medical directors of mass radiography units. One of the most surprising things in reading large series of miniature films is the extraordinary variation in the size, shape, and position of the cardiac silhouette: most of these cases are regarded as within normal limits, but numerous examples of considerable enlargement and abnormality of the cardiac outline are encountered.

In the past, cases regarded as grossly abnormal were recalled and examined by the medical director, who submitted a report to the patient's doctor. It has been felt, however, that this procedure was inadequate, and recently we have established a liaison with the cardiovascular unit in the Newcastle General Hospital, where these cases can be adequately investigated. In all cases a report is sent from the cardiovascular clinic to the patient's doctor, with a copy to the medical director of the mass radiography unit. In cases of dextrocardia where the abnormality was not known a letter is sent to the volunteer in order to show it has not been overlooked.—I am, etc.,

Newcastle-upon-Tyne. W. H. DICKINSON.

Atelectasis and Bronchiectasis

SIR,—I was interested to learn that Dr. B. Mann (December 9, p. 1331) had found in cases of "reversible" bronchiectasis that the intrapleural pressure was not appreciably lowered, contrary to what would have been expected from a perusal of Lander and Davidson's (1938) paper. I also observed this puzzling feature not only in cases of "reversible" bronchiectasis, but in permanent atelectatic bronchiectasis as well. I hope shortly to publish an account of work on this subject, but briefly I came to the following conclusions:

The essence of Lander and Davidson's important article was that, when pulmonary collapse due to bronchiolar occlusion occurs, there is a loss of space in the affected hemithorax, and to compensate for this there is compensatory emphysema and a compensatory dilatation of the bronchi in the collapsed portion of lung. Intrapleural pressures are readings of the gas pressures in a small artificial pneumothorax, and give a measure of the *mean* elastic tension of the lung. It occurred to me that if, following collapse of a portion of the lung, the resulting increase in elastic tension were largely localized to the affected part, the contained bronchi might be dilated without the intrapleural pressure, which measures the *mean* tension of the lung, being necessarily markedly lowered, as Lander and Davidson believed it was. I later read the excellent article by Andrus (1937) in which he stated that following pulmonary collapse the increased lung stresses are in fact largely localized to the affected area. Therefore, though I do not believe that a low intrapleural pressure has anything to do with the production of bronchiectasis, or is even a necessary accompaniment of atelectatic bronchiectasis, I see no reason to doubt the essential features of Lander and Davidson's work.

Artificial pneumothorax was employed on an experimental basis, and not solely for its therapeutic effect. The affected bronchi were not constricted as Dr. Mann feared. The lung was relaxed until the calibre of the dilated bronchi had returned approximately to normal as shown by bronchography. This was thought to be adequate to allow escape of secretion; otherwise one would have to suppose that, the more marked the bronchiectasis in a collapsed lobe, the better the prospects of re-expansion.

I have no intention of generalizing from the results of my series of pertussis cases, but I was most interested to find that Dr. Franklin (December 9, 1950, p. 1331) has noted a similar low incidence of residual respiratory trouble. I entirely agree with him that it is unsatisfactory to blame pertussis for bronchiectasis on the strength of a history taken often years after the beginning of symptoms; at the same time I have encountered a few cases, out of scores, in which the connexion is too clear cut to be seriously doubted. Experience of bronchiectasis convinces me that Dr. Franklin is correct when he says that the prognosis of bronchiectasis in childhood is much better than is commonly stated. Nevertheless I still think that there are unilateral cases in which the affection will be at best a recurring nuisance, at worst a disabling affliction, and in which surgery holds out the only reasonable prospect of complete cure.—I am, etc.,

Glasgow. A. W. LEES.

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Lung Abscess after Electro-convulsive Therapy

SIR,—I have read with interest your annotation (December 16, p. 1377), but I think that it would be unwise to assume that a 1% incidence of this complication is universal. Pulmonary abscess is indeed a serious complication, and it seems strange that if one case in every hundred is liable to be affected so little attention has previously been focused on it. Conway and Osmond (1948) report one case, and quote Kalinowski (1946) as stating that there is only one other case in the literature. In his comprehensive review of shock therapy Wilcox (1948) states that "pulmonary abscess has been a complication in some clinics," but cites no references in British or American literature. In articles dealing with the technique, complications, and results of electro-convulsive therapy (E.C.T.), Palmer (1948) and Dax (1949) make no mention of this specific complication.

In over 5,000 cases treated by E.C.T.—i.e., double the number quoted by Kwalwasser *et al.*—I have encountered an abscess in only one patient, and even in this case it was not possible to attribute it entirely to E.C.T., as the patient had had a dental extraction under general anaesthesia a week before admission to hospital. Almost without exception, every case in this series was followed up for at least a year. Atropine was not used, but the patient was turned on his left side as soon as the clonic stage terminated. Temperature charts were kept, and, if any suspicious rise was noted or any respiratory symptoms exhibited, the patient was seen by the consultant physician and the chest *x*-rayed.

There seems little doubt that the incidence of lung complications during or following E.C.T., insulin shock therapy, or

prolonged narcosis is inversely proportional to the standard of nursing. In one mental hospital in this country it was found that, following these treatments, pneumonia had occurred in six men, whereas no such complication had arisen during the same period in treating twice as many women. When specially trained male nurses were allocated to the centre the incidence of pulmonary complications fell rapidly.

As the last paragraph of your annotation raises much wider issues, may I as one who has used E.C.T. fairly extensively since its inception in this country be permitted to express my personal views? On the basis of experience in treating a large number of patients, I feel that out-patient treatment should be given only in exceptional cases, and that the practice of admitting patients to hospital for E.C.T. and sending them home in a fortnight or so is to be deprecated. Even when few treatments are given, a mild cumulative confusional state may occur during the early convalescence period. The patient is perplexed and worried for a day or two, but if medical reassurance is not immediately available a relapse often results. It is well worth while to retain patients in hospital for at least three weeks after the last electrical treatment has been given. The highly charged emotional aura surrounding E.C.T. has in the past prevented a balanced and unbiased general evaluation of this form of therapy. The late Dr. C. C. Burlingame (1947), that notable advocate of tolerance and mutual understanding among psychiatrists, has, however, summed up the situation as follows: "Only a stupid person would decry shock therapy as an instrument in helping the mentally ill . . ." but "to proceed as if shock therapy, any more than any other psychiatric technique, can usurp the place of all other therapies is psychiatric nonsense."—I am, etc.,

Morpeth. W. LIDDELL MILLIGAN.

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Treating the Ruptured Uterus

SIR,—I have read and re-read the article by Dr. A. H. C. Walker (November 25, p. 1205) describing his method of dealing with a ruptured uterus occurring in labour. It appears that he inserted his hand through the rupture, carried out a podalic version in the abdominal cavity, and then dragged out foetus and placenta through the vagina. A vaginal hysterectomy was then performed, and the patient died after three days.

Dr. Walker states that he carried out these procedures because he suspected that the uterine artery had been ruptured, and that the patient could not survive until theatre facilities were available—a matter of two hours. Here I disagree with him. It was clear that the uterus had ruptured some three hours before Dr. Walker saw the patient—i.e., when the pains suddenly ceased. In rupture of the lower segment, bleeding occurs from torn sinuses and occasionally from the uterine vein, but rarely from tearing of the uterine artery; and, if any sizeable vessel is torn, then bleeding is profuse, the systolic pressure falls to 50 to 60 mm. Hg in about half an hour, and the patient is exsanguinated within one and a half to two hours unless the bleeding has been dealt with surgically. This is in contrast with rupture in the upper segment through the scar of a classical caesarean section, in which case the uterus acts as its own haemostat when its contents have been extruded into the abdominal cavity and time can be spent in resuscitation.

In the case reported the blood pressure was 80/40 three hours after rupture; therefore she had obviously not ruptured any major vessel but was suffering more from peritoneal shock than from actual blood loss. If rupture of the uterus had been diagnosed from the history and the abdominal findings, and vaginal examination had been avoided, then the patient could have been safely tided over with morphine for the next two hours, and the usual abdominal delivery of the foetus and placenta carried out followed by a subtotal hysterectomy. I assume that plasma is available in Ibadan.—I am, etc.,

Church Village, near Pontypridd. ROBERT HODKINSON.

Manubrio-sternal Joint in Rheumatoid Arthritis

SIR,—Experience with Dr. W. S. Tegner's patients in the Department of Physical Medicine of the London Hospital confirms the views of Drs. A. Bogdan and J. Clark (December 16, p. 1361) and supports the statement by J. J. Conybeare that "in rheumatoid arthritis any joint may be affected."

Since September, 1949, 11 cases have been seen of involvement of the manubrio-sternal joint in patients known to be suffering from rheumatoid arthritis. Of these only one case occurred in a man. There appeared to be a seasonal incidence, as two cases were first observed in September, three in October, and two in November, 1949, two in January, one in April, and one in May, 1950.

As the authors point out, the pain has to be distinguished from other causes of chest pain, and one of our early cases had been submitted to cardiovascular investigations, and a diaphragmatic hernia had been considered. In this region not all swellings which are tender are necessarily due to rheumatoid arthritis, and occasionally in patients with juxta-articular panniculitis a painful tender fatty swelling may be found lying over the manubrio-sternal joint.

Two further points of interest were noted in our cases. Seven out of the 11 patients gave a history of cough; and five of the patients also had involvement of certain of the costochondral junctions, with local pain and tenderness and swelling. In three cases the left third costochondral junction was principally affected. In another young woman with active rheumatoid arthritis but with no involvement clinically of the manubrio-sternal joint there developed such a marked swelling and tenderness of the left third costochondral junction that the patient was afraid she had a growth and was only reassured when the swelling subsided in about seven weeks.

—I am, etc.,

London, E.1. M. Q. BIRKBECK.

Registrars

SIR,—Why has no one stated in the controversy about registrars that the vast majority of these jobs were simply created in order to provide experience for young doctors who had grown rusty in the Forces? The appointments were made, not to meet the need of the hospitals, but simply to remedy rustiness. Surely, therefore, many of them ought to be abolished five years after the war? The appointments of house-surgeon and house-physician will then be more instructive and responsible.—I am, etc.,

Colchester. M. E. LAMPARD.

SIR,—Only a short time ago (although long enough for us all to realize the truth of what was said at the time) your leading article wisely pointed out—one might almost say pontificated—the unsatisfactory terms of the National Health Service both in administration and in the doctors' role. Wise letters also filled your columns on the matter, but nevertheless as the appointed day drew near a sudden scurry for cover was made by all but a very few who remained outside the Service. How history repeats itself. Once again your leaders pontificate on the dastardly behaviour of the Minister with regard to the registrars, and wise letters appear. For the registrars' encouragement I should like to assure them that unless firm, coherent, cohesive, and coercive action on their part is forthcoming 1,100 of them will find themselves in the street very soon. I would further warn that unless the consultants choose to support them they will find themselves facing a similar unpleasant shock in about six months' time, when they will probably receive the same degree of comforting editorials and letters.—I am, etc.,

Burton-on-Trent. R. LUNT.

Obituary

Sir PHILIP PANTON, M.B., B.Chir.

Sir Philip Panton, consulting pathologist to the Ministry of Health, died at Bourne End on December 27.

Philip Noël Panton was born at Wareham, Dorset, and was educated at Harrow; Trinity College, Cambridge; and St. Thomas's Hospital. He qualified in 1903 and graduated M.B., B.Chir. a year later. After holding the posts of house-physician at St. Thomas's and house-surgeon at Addenbrooke's Hospital he obtained a Louis Jenner research scholarship in pathology at his own old teaching school and a Gibson research scholarship at the Society of Apothecaries. He was then appointed assistant director of the clinical laboratories at St. Thomas's, but shortly afterwards joined the staff of the London Hospital, where for many years he was clinical pathologist and director of laboratories. He published a number of important papers and wrote the well-known textbook *Clinical Pathology*, the authorship of which he shared in later editions with Professor J. R. Marrack. During and after the second world war Panton advised the Ministry of Health on the establishment of pathological services to meet the needs of the E.M.S., and the detailed knowledge he gained as he went about the country served him in good stead when plans had to be made for the National Health Service. Many medical men, as well as laymen concerned with hospital administration, must have met Sir Philip Panton on his visits to hospitals and laboratories and been impressed by his cultured urbanity and his attention to detail even at the end of a long and tiring day. He served on several important committees of the B.M.A., including the Central Emergency Committee, the Central Medical War Committee, and the Postgraduate Education Committee. For his services to the Ministry of Health Panton was knighted in 1946.

Walter Stoneman, London.

Professor J. R. Marrack writes: I have seen the growth of the clinical laboratory of the London Hospital under Sir Philip Panton's direction from 1909 until he left in 1946. He had found very haphazard arrangements when he came, but he quickly had organized a laboratory that was able to cope with any demands that clinical medicine made at that time. Under his guidance it has continued to meet the ever-growing demands and to undertake research over a wide field. Sir Philip's interest was in medicine as a whole; laboratory work to him was an aid in the understanding and diagnosis of disease and the treatment of patients. His book *Clinical Pathology*, which first appeared in 1913, showed his outlook; it did not, as did most books of that time, include a multiplicity of tests good and bad, but only those that he had found useful. It was compiled almost wholly from his own experience—written, as he told me, in bed in the early hours of the morning.

In those days, with a less crowded course, students who wished to could take a post as clerk in the clinical laboratory. I took this post in the hot summer of 1911. Panton made those three months most interesting and valuable. Like many others, I have owed a great deal both then and in the years after to his interest and encouragement. He was unusually able to appreciate other people's points of view in scientific matters and to realize the value and importance of work that was not in his line and had no immediate application in diagnosis or treatment. This and his general interest in medicine made him an excellent intermediary between the more specialized laboratory workers and clinicians and between medical men and laymen.

Sir LEONARD PARSONS, M.D., F.R.C.P., F.R.C.O.G., F.R.S.

Professor Hilda Lloyd, P.R.C.O.G., writes: Sir Leonard Parsons's interests in the medical world were so wide that there is no branch of medicine that will not feel his loss. His contributions to paediatrics were so well recognized by the Royal College of Obstetricians and Gynaecologists that he was admitted to the Fellowship of the College in 1944, and at that time delivered an outstanding lecture on antenatal paediatrics. His interest in the care of the newly born during the time he was in charge of them at the Maternity Hospital, Birmingham, laid the foundation upon which the present department has been built up. Although his tastes in medicine were catholic, perhaps none of them were of greater worth than this, his own particular choice. He had an uncommon facility for picking out young promising juniors, and many owe a great deal of their success to his help and encouragement in the early days of their careers. So passes a great friend of youth and, not unnaturally, one who retained his youthful outlook in ripening age.

J. C. S. writes: Leonard Parsons's life was like a race well run. Quietly, steadfastly, and modestly it went on from success to success. He saw early that paediatrics could make great contributions to the teaching and practice of medicine, that it was one of the bulwarks against excessive specialism, and he designed his career to those ends. In one of his rare but delightful moments of self-confession he told me that it was on a return journey from America, where he had learned so much, that he saw how an English hospital might design its work to combine research, teaching, and practice in an atmosphere of comradeship. Those who worked with him in Birmingham will know best how quickly he did this to make his hospital one of the most famous and happy paediatric centres in the world. But it was obvious to all of us who knew him that the success of his designs was achieved as much through the warmth of his charity as through the eagerness of his mind. He was truly both a great physician and a beloved man. In 25 years of ripening friendship I never heard him speak ill of any man, nor any man say ill of him. In these ways he went on gathering fame, and always a little surprised that it came as it did. So many memories of Parsons crowd the mind that it is difficult to choose, but I shall cherish always the spontaneous kindness of his remarks to a younger man at the last meeting of the British Paediatric Association. In that frankness of discussion which has characterized the Windermere meetings the young man had been somewhat roughly handled, and Parsons sprang to his defence with words of praise, advice, and encouragement that enriched the day. He endured unfalteringly, so that none of us could believe

his age. It was therefore fitting, though tragic, that his life should end suddenly on a Sunday morning when he came home from his chapel, warmed himself at the fire, and died.

HENRY MacCORMAC, C.B.E., M.D., F.R.C.P.

Dr. G. N. Meachen writes: I had not long been appointed to the staff of the Blackfriars Skin Hospital (then in the old premises in Stamford Street) when a tall, pleasant-looking young man came one afternoon to my clinic and asked, somewhat diffidently, if he might be allowed to sit with me to "see some skins." I quickly discovered that here was no casual postgraduate hoping to pick up a few crumbs of dermatological knowledge but a real student of what was then almost a neglected specialty, anxious and willing to learn all that he could. It was not long before I felt sufficient confidence in my new clinical assistant to leave him in charge of an occasional clinic. Thereafter I watched his upward progress with the greatest interest, and I rejoiced to see him mount the staff ladder at the Middlesex Hospital. He was indeed a "loyal and lovable friend."

L. K. writes:

> "Steel true and blade straight,
> The Great Artificer made my mate."

Henry MacCormac and I were close friends before the first world war, and it was sheer delight to me at Wimereux, where I was in charge of a hospital in 1914, to find that he was establishing a skin hospital at Hardelot-Plage some 20 miles away. His work there was almost superhuman and was greatly praised, especially as he was able to send up to the front in a short time many hundreds of patients who would otherwise have had to come to England to be treated. He will be much missed, for not only was he a brilliant dermatologist but a most amusing companion, sparkling with that Irish humour which is inborn and cannot be acquired. He certainly had the courage of his convictions, and his great enthusiasm to carry a point was always admired. His quiet personality was irresistible, and his smile at times insidiously enchanting: he was indeed a lovable gentleman.

Dr. ALEXANDER DALRYMPLE MCQUEEN, who was for many years in practice in Blackheath, Birmingham, died at his home in Birmingham on November 23, aged 74 years. McQueen entered Aberdeen University at the age of 16, and graduated M.B., Ch.B. in 1898. Before establishing himself at Blackheath, where he remained for 50 years, he practised for a short time in Oldhill, Staffordshire. He lived to see very great advances in medicine and surgery, and could recollect when major surgery was performed in the homes of the patients. He was interested in the work of the St. John Ambulance Brigade, and was the first divisional surgeon and lecturer to the Rowley Regis Division. Well known as an examiner at competitions in the West Midlands, he was recently awarded the long-service medal. He was a skilful and conscientious practitioner to whom the welfare of his patients was his only concern. His somewhat austere manner cloaked great kindliness and benevolence. The "Black Country" has lost one of its oldest and most able practitioners, and he will be greatly missed in the district where he worked for so many years. Dr. McQueen leaves a widow and three daughters.—J. R.

Dr. TIMOTHY GERRARD DEMPSEY died in Dublin on November 25, 1950. He graduated M.B., B.Ch. at the National University of Ireland in 1941, and held various hospital appointments until 1943, when he joined the staff of the Grove Park Hospital, London. It was here that his interests became centred on tuberculosis, and he quickly showed an aptitude in the field of clinical research. This led to his being associated with the first clinical trials in Britain of P.A.S., and it is in connexion with this form of therapy that his name is best known. Thereafter he devoted most of his time and energies to the study of this new weapon in the fight against tuberculosis. He was invited to visit the Continent, America, and India, where he delivered addresses on his experience with P.A.S. In 1948 he joined the staff of the Mayday Hospital, Croydon. Here, with his special knowledge of the chemotherapy of pulmonary tuberculosis, he instituted a new regime of treatment, and inspired his co-workers with fresh enthusiasm in their difficult task. He never spared himself at any time in his efforts for the welfare of his patients and constantly overtaxed his own limited physical strength on their behalf. Finally his own health broke down in November, 1949. During long months of illness he never lost heart, and his own keen interest in medicine did not diminish. His characteristic humour never failed him, and when he knew the end was in sight his courage was an inspiration to all. His charm and sympathetic manner endeared him to his patients and colleagues, and his death at the early age of 34 is a loss to the medical profession as a whole and the tuberculosis field in particular. His many friends will wish to express their deep sympathy with his widow and two young children.—L. M. K.

Dr. GILBERT JOHN KING MARTYN, consulting physician to the Royal Mineral Water Hospital, Bath, died at Paignton on December 5, aged 81 years. He studied medicine at Cambridge and the Middlesex Hospital, graduating M.B., B.Ch. in 1894. Three years later he proceeded M.D., and also took the D.P.H. After qualifying, he became a house-physician at his own hospital, and was later resident medical officer at Queen Charlotte's Hospital. King Martyn went to Bath in 1898 and was in partnership for a short time with Dr. S. P. Budd, who died the following year. For nearly 25 years he carried on a large residential and spa practice. In 1913 he was appointed physician to the Royal Mineral Water Hospital, a post that he held until his retirement in 1922. During the first world war he was so over-worked that his health broke down, and, although it was completely restored by a sojourn in the Balearic Isles, he returned to Bath for a short period only, and finally retired from practice in 1922, when he went to live at Draguignan in France. King Martyn was a member of the B.M.A., and was president of the Bath and Bristol Division in 1920–1. One of the eight founders of the Bath Clinical Society in 1908, he became its president in 1913. He was a keen fisherman, and was interested in the arts, especially painting and music. Of distinguished presence and courteous manners, he was popular with both his colleagues and his patients. He was twice married, his first wife dying about 30 years ago.—R. W.

Dr. ARTHUR EDWIN RAINE, medical superintendent of Bishop Auckland General Hospital and part-time assistant senior administrative medical officer to the Newcastle-upon-Tyne Regional Hospital Board, died suddenly on December 11 in the hospital for which he had done so much since July, 1948. He was the son of R. W. Raine, J.P., of Middleton-in-Teesdale, and he studied medicine at Newcastle-upon-Tyne, graduating M.B., B.S. in 1916 after a distinguished academic career, in which he obtained honours in all his professional examinations. Soon after graduation he was on active service in Salonika, and in 1918 he was invalided out of the R.A.M.C. suffering from malarial fever. After this he spent a few years in Sunderland in general practice—an excellent preparation for his future career with the Durham County Council and with the Newcastle-upon-Tyne Regional Hospital Board. For 23 years he gave able and devoted service to the county

medical officer in charge of its public assistance medical services. After the National Health Service Act came into force in 1948 Raine had two outstanding professional interests. In his capacity as medical superintendent of Bishop Auckland General Hospital he did more than any other individual to turn this hospital, within that short time, from a poor-law institution and infirmary into an active and well-staffed general hospital of about 300 beds which is already dealing with a major share of the acute hospital work of the 100,000 people who live in and around Bishop Auckland. It was a great joy to Dr. Raine to see this rapid development and to have the Bishop Auckland General Hospital recognized by the General Nursing Council as a complete general training school for nurses. Many of his plans must now be completed by others, but his wisdom has assured the future success of the hospital which held such a strong place in his affections. Raine was proud of his unique appointment as part-time assistant senior administrative medical officer to the regional hospital board and was grateful for the opportunity which it gave him to continue his service for the whole of County Durham. His wisdom and knowledge were of the highest value to the board in the planning of the hospital service, for his complete and intimate knowledge of the county and his friendship with innumerable people in it gave to the board in its early years something which no other doctor in the region could have given. His own clinical interests were in the care of the aged sick, and in this clinical field he was developing a good service in Bishop Auckland and expected to see much more accomplished in the next few years. In addition to his other onerous duties Raine was also one of the original members of the Bishop Auckland Hospital Management Committee, which owed to him a great debt for his wise guidance in the development of the other hospitals in the group. He was a man of great physical and mental energy who did not spare himself in the course of his duties, but who found recreation in music and happiness in his church. His severe illness a few weeks ago was therefore a great surprise to his colleagues, and his sudden death, when he appeared to be getting over the illness, was a great shock. Dr. Raine thought seldom of himself and always of others, especially the aged sick, and his friends and colleagues sympathize with his widow and his two brothers and two sisters in their loss.—W. G. P.

Dr. JOHN RUSSELL LOWE, who died at sea on December 12 while medical officer of the *City of Chester*, was a graduate of Trinity College, Dublin, and was himself the son of a doctor. A good sportsman, he had played rugby for his school, Campbell College, Belfast, and his university, and he had also rowed for Trinity. He was only 28 years of age when he died, having been qualified four years. In all his house appointments he not only won for himself the warm esteem of his chiefs but the very real gratitude of his patients. His loyalty and generosity to his friends were, perhaps, his most outstanding qualities. Only those who knew him well learned how genuine and unostentatious were his many acts of kindness. His tragic death at the outset of a most promising career comes as a great shock to his unusually many friends.—R. W. M. S.

G. B. writes: A member of a family who owe much to the skill and care of Dr. Robert Young Aitken (whose obituary notice appeared in the *Journal* of November 4, 1950) would like to record that many patients will remember him no less for his kindness and generosity—qualities often hidden by a gruffness of manner. In remembering him as he went about Blackburn and the surrounding district in those hard-pressed days during and after the first world war, a vivid picture springs to mind of the tall, alert Scot leaping from car to door and back again. For those in real need, Dr. Aitken put himself to endless trouble. There must be many who will think of him with gratitude.

Medico-Legal

SURGEON FINALLY CLEARED OF NEGLIGENCE

[FROM OUR MEDICO-LEGAL CORRESPONDENT]

In 1942 Mr. J. F. Whiteford, an American consulting engineer of 65, was declared by Mr. John Hunter to be suffering from cancer of the base of the bladder. On the strength of this diagnosis he returned to America, gave up his practice, put his affairs in order, and prepared to die, but Dr. Benjamin Barringer, an American surgeon who saw him several months later, found that his disease was not in fact malignant. Mr. Whiteford accordingly sued Mr. Hunter for damages, alleging negligence in not using a cystoscope or performing a biopsy. Mr. Justice Birkett, trying the case at first instance in 1948, awarded heavy damages, but the Court of Appeal next year reversed this judgment and gave Mr. Whiteford leave to appeal to the House of Lords. Both hearings were fully reported, and some comment on the case was made in a leading article in this *Journal*.[1] Their lordships' House has now given judgment dismissing Mr. Whiteford's appeal.[2]

Lord Porter stated in his opinion that Mr. Whiteford's argument was not that the diagnosis was negligent but that the resemblance between real and simulated cancer was so close that no diagnosis of cancer should be accepted without a pathological examination. A surgeon must exercise the degree of skill and care reasonable for one in his position, and if charged with negligence could clear himself by showing that he had acted in accordance with general and approved practice. Mr. Hunter had given evidence, supported by two eminent surgeons, that his action had conformed to the skilled practice of the profession. On the other hand Dr. Barringer had said that to verify a diagnosis of a cancerous growth a complete examination by means of a cystoscope and through the open bladder, and a pathological examination of any questionable area, should have been made, that he took those steps himself, that they were the ordinary and usual steps, and that there was no difficulty or danger in either operation.

Lord Porter, however, did not think Dr. Barringer meant that Mr. Hunter should have taken those steps in March or April. The appearance and state of the bladder at that time did not seem to have been disclosed to Dr. Barringer, whose answers did not suggest that he had in mind the "indurated mass covered with thickened mucous membrane abnormal in colour" described by Mr. Hunter, but a condition like the very different condition he himself had found in New York. Moreover, even if his evidence could be accepted in full, a specimen could have been taken only with a cystoscope fitted with a rongeur attachment, an instrument very rare in England in 1942 and not possessed by Mr. Hunter. All the English medical witnesses had asserted that it was against approved practice in England to use a cystoscope where there was acute urinary retention, and that when the bladder was drained and collapsed it was difficult or impossible to use effectively a cystoscope not of the flushing type. This instrument also had been rare in England at the material time, and Mr. Hunter had not possessed one. The appeal was dismissed by unanimous vote of their lordships.

[1] 1948, **2**, 537; 1949, **1**, 595, 669.
[2] *The Times*, November 17, 1950.

"Persons without a settled way of living" (vagrants or casuals) in December, 1949, averaged 2,367 in reception centres of the Ministry of National Insurance. This was the first time since the war that there has been a decrease, though all the post-war figures are low compared with formerly.

Universities and Colleges

UNIVERSITY OF CAMBRIDGE

In Congregation on December 16, 1950, it was decided to establish a University Lectureship in the Department of Medicine.

The following degrees were conferred on December 16, 1950:

M.A.—W. J. St. E.-G. Rhys, M.B., B.S., Janet E. Bottomley, M.D., F.R.C.S., M.R.C.O.G.

The following candidates have been approved at the examination indicated:

FINAL M.B.—[1]R. H. Adrian, [2 3]W. V. Agnew, [1]P. T. Annesley, [1 2]V. Armett, [1 2]L. J. H. Arthur, [1 2]N. B. Atkin, [1]R. S. Atkinson, [1]A. M. Baker, [1 2]J. A. C. Bamford, [1]G. S. Banwell, [1 2]A. P. Barclay, [3 4]M. E. Barton, [1]A. C. Beatty, [1]R. J. Bennison, [3 4]A. P. Blower, [3 4]M. H. S. Bound, [1 2]J. W. Bradbeer, [1 2]S. D. Bradley, [1]M. Braimbridge, [1]J. S. N. Briggs, [1 2]G. S. Brindley, [3 4]P. L. Broadhead, [2 3]I. M. Brown, [3 4]W. M. Browne, [3 4]K. Brown-Grant, [3 4]A. A. F. Bryson, [1 2 3 4]W. E. G. Buckley, [3 4]D. M. J. Burns, [1]N. P. Burns, [1 2]A. S. F. Butcher, [1 2]E. P. Cadbury, [3 4]M. D. Cameron, [2]C. McG. Campbell, [3 4]P. B. Clark, [1 2]E. H. Coats, [1 2]R. A. Cooper, [1 2 3 4]P. H. Crosskey, [1 2]J. C. M. Currie, [3 4]W. G. Davies, [3 4]D. G. Dickson, [3 4]J. H. Dickson, [1 2]J. M. H. Dickson, [3 4]W. Dorrell, [3 4]G. D. Downes, [1]M. F. Downey, [1 2]B. O. L. Duke, [2]F. P. D. Easby, [1 2]J. Fairgrieve, [1 2 3]R. T. D. FitzGerald, [1 2]R. M. F. Fox, [1]R. T. Gaukroger, [3 4]A. E. Gibbs, [3 4]D. C. Gibbs, [3]P. C. Glynn-Jones, [1]L. Golberg, [3 4]P. Goodall, [1 3]P. H. Gorodi, [3 4]H. Gough-Thomas, [1]D. M. Grant, [1]I. H. Grant, [1 3 4]V. A. Grimshaw, [1 2]I. M. Guiver, [1 2]A. T. G. Gunn, [1 2 4]B. M. H. Hardman, [1]A. M. Harington, [3 4]A. S. Harris, [3 4]D. S. Harry, [3 4]G. Harvey Evers, [3 4]M. W. A. Haward, [3 4]J. M. Heaton, [3 4]J. F. Higgins, [1 2]U. M. P. Hill, [1 2 3 4]G. Hirst, [3 4]M. Hobsley, [3 4]C. A. Holborow, [1 2]M. L. House, [3 4]R. L. Huckstep, [1 3]P. E. Huddy, [1]R. G. Huntsman, [3 4]J. Hurst, [1]A. W. Johnston, [3 4]E. W. Jones, [1 2]W. R. Juckes, [1]R. Keeley, [1]J. C. Kelsey, [3 4]M. H. King, [1]R. G. P. King, [3 4]I. Kinross, [1 3]C. D. Lacey, [1 2]A. B. McGrigor, [1 2]K. G. P. Mackenzie, [1]R. E. Mackie, [1 2]J. J. McMullan, [1]C. R. Maddock, [3 4]V. J. Marmery, [1]S. E. Marmion, [1]Mrs. C. M. Maxwell, [3 4]I. H. Mills, [1]K. H. G. Milne, [3 4]J. F. Moor, [3 4]D. B. Murray, [2 3]J. H. Newton, [2]G. McK. J. Nicholl, [3 4]D. S. H. W. Nicol, [2]E. M. Orton, [3 4]A. Paneth, [3 4]G. G. K. Parsons, [1 2]F. S. Perry, [1 2]J. R. H. Pinkerton, [1 2]H. F. W. Pribram, [3 4]E. J. Purcell, [1 2]P. J. Randle, [1 2]J. R. Rees, [3 4]R. H. Reynolds, [3 4]J. M. G. Ritchie, [1 2]H. M. Roseveare, [3 4]H. B. Russell, [3 4]F. C. Rutter, [1 2]J. J. H. Rymer, [3 4]H. A. Sanford, [1 2]G. E. Sayce, [3 4]J. R. Seale, [3]J. H. Sewart, [3 4]C. Shaldon, [3 4]Mrs. E. C. Shore, [3 4]P. H. Shorthouse, [1 2]I. M. Simmonds, [1 2 3 4]H. S.-K. Singha, [3 4]G. W. Smallcombe, [3 4]D. W. Smith, [1]I. Smith, [1]J. L. Somervell, [3 4]E. M. Sproston, [3 4]C. M. Stafford, [3 4]P. C. Steel, [2 3 4]E. Stokoe, [3 4]J. D. E. Strong, [3 4]H. Sunderland, [3 4]P. H. Swinhoe, [1]G. A. Taylor, [2]K. A. Taylor, [1 2]B. E. L. Thompson, [3 4]G. S. Thompson, [1 2 3 4]P. W. Tucker, [1]B. V. Tyman, [3 4]I. M. Watts, [1]W. G. Wenley, [3 4]Mrs. J. M. Weston, [1 2]P. H. Wheeler, [3 4]Mrs. M. P. Whitehouse, [3 4]I. H. Williams, [1]J. A. Williams, [2 3 4]R. A. Williams, [1]R. T. Williams, [1 2 3]D. H. M. Woollam, [1 2 3]G. H. Wright.

[1]Passed in pathology and pharmacology. [2]Passed in principles and practice of physic. [3]Passed in principles and practice of surgery. [4]Passed in midwifery and gynaecology.

UNIVERSITY OF EDINBURGH

At a Graduation Ceremonial held on December 15, 1950, the following medical degrees and diplomas were conferred:

M.D.—E. D. Caldwell, T. M. Chalmers, E. N. Moyes, [1]C. S. Russell, [2]J. T. R. Russell, [1]J. J. Stevenson.

M.B., CH.B.—H. F. Acheson, W. D. A. Birch, G. Y. Cheyne, Phyllis Connor, B. S. Dorfman, I. A. Fraser, A. D. Gentle, J. Grant, E. R. Henry, Brenda M. Johnson, Hazel M. Lazarus, Margaret E. Loudon, A. Macaulay, A. R. Manners, W. P. Mazur, J. A. Mitchell, P. T. Monard, R. Renton, P. F. F. Rushforth, T. M. N. Salvesen, L. E. S. Sandison, G. A. Steele, J. F. Stephen, E. B. Tagoe, Nancy M. Tweedie-Stodart, Mary B. Watson, J. S. Whittaker, T. W. Wilkinson.

DIPLOMA IN MEDICAL RADIOTHERAPY.—J. D. Abbatt.

The following awards were made: *Crichton Research Scholarship*, A. D. Robertson, M.B., Ch.B. *Ethicon Research Fellowship*, S. R. Mukherjee, M.B., B.S. *George Guthrie Research Fellowship in Child Health*, H. S. Provis, M.B., Ch.B. *Hastilow Research Scholarship in Rheumatism*, B. Cruickshank, M.B., Ch.B. *Paterson Travelling Scholarship in Surgery*, A. B. Wallace, M.B., Ch.B. *Andrew Graham Ritchie Bursaries*, D. B. Eaton and N. L. M. Browne. *Russell-Crighton Prize*, K. Wood.

[1]In absentia. [2]Commended for thesis.

UNIVERSITY OF DUBLIN

SCHOOL OF PHYSIC, TRINITY COLLEGE

The following candidates have been approved at the examinations indicated:

M.D.—R. W. Barter, M. T. S. Conradie, B. V. Earle, H. FitzGibbon, J. M. Johnston.

M.A.O.—C. L. S. Archer, M. B Flanagan.

M.B.—*L. Tarlo, P. J. Myles, T. H. Bewley, A. C. Allen, H. A. Devlin, R. W. Mitchell, J. P. B. Tuohy, R. S. W. McLarnon, D. L. Cowan, N. Furlong, Sara K. Mitchell, D. D. Macdonald, N. R. McNutt.

B.CH.—*A. T. F. ffrench O'Carroll, Helen M. Hackman, P. E. A. Blake Knox, Anna J. Hendly, M. J. Sweeney, J. N. Cockrill, J. O. Hunter, A. W. L. Pollock, A. Rosenthal, F. H. Moore, E. W. Lillie, R W. Mitchell, L. Tarlo, D. Romney, W. G. Hutchinson, D. D. Macdonald, J. P. B. Tuohy.

B.A.O.—*Grace M. F. Yorke, *R, V. Lea, *W. McC. Wilson, H. J. Neethling, Sara K. Mitchell, A. W. L. Pollock, N. R McNutt.

The following medical degrees were conferred on December 7, 1950:

M.D.—R. W. Barter, H. FitzGibbon, J. M. Johnston.

M.A.O.—C. L. S. Archer, M. B. Flanagan.

M.B., B.CH., B.A.O.—A. C. Allen, T. H. Bewley, J. N. Cockrill, H. A. Devlin, N. Furlong, Helen M. Hackman, Anna J. Hendly, J. O. Hunter, W. G. Hutchinson, P. E. A. Blake Knox, R. V. Lea, E. W. Lillie, D. D. Macdonald, N. R. McNutt, Sara K. Mitchell, F. H. Moore, H. J. Neethling, A. T. F. ffrench O'Carroll, D. Romney, A. Ross, M. J. Sweeney, J. P. B. Tuohy, Grace M. F. Yorke.

*With second-class honours.

UNIVERSITY OF DURHAM

In Congregation on December 15, 1950, the following medical degrees and diplomas were conferred:

M.B., B.S.—Meryl A. Christian, J. A. Eykelenboom, J. R. J. Glavina, C. St. J. E. Harding, Marjorie Hepple, D. D. Hobbs, Shirley McTaggart, B. Metcalfe, Shirley M. C. Nuttall, Lola V. Stafford, Anne W. Watts.

D.P.H.—J. F. McGarity (*in absentia*).

Henry Harvey Evers, M.S., F.R.C.S., Lecturer in Midwifery and Gynaecology in the University, has been appointed to the Chair of Midwifery and Gynaecology at Newcastle-upon-Tyne.

UNIVERSITY OF LONDON

Dr. Vernon Frederick Hall has been appointed Dean of King's College Hospital Medical School as from January 1, in succession to Mr. Harold C. Edwards, who has resigned, and Mr. Alexander John Heriot has been appointed Vice-Dean.

Mr. F. Bergel, Ph.D., D.Sc., will deliver two lectures in the physiology theatre, University College, Gower Street, W.C., on Thursdays, January 18 and 25, at 5.15 p.m. His subject is "Physico-chemical Properties and Pharmacological Activity of Drugs."

Two lectures on "Cell Metabolism and Chemotherapeutic Activity" will be delivered by Mr. T. S. Work, Ph.D., in the physiology theatre of University College on Fridays, February 2 and 9, at 5.15 p.m.

All the above lectures are open to members of the public.

Mr. W. J. Corlett will deliver a lecture on "Population Trends" in the anatomy theatre, University College, Gower Street, W.C., on Thursday, February 15, at 1.15 p.m.

A lecture on "Counting Heads—The Taking of the Census. 1801–1951" will be delivered by Mr. A. J. Taylor in the anatomy theatre of University College on Thursday, March 1, at 1.15 p.m.

Dr. E. Ashworth Underwood will deliver a lecture on "John Wesley and his 'Primitive Physick'" in the anatomy theatre of University College on Thursday, March 8, at 1.15 p.m.

Admission to the above lunch-time lectures is free, without ticket.

The title of Professor of Clinical Pathology in the University has been conferred on Montague Maizels, M.D., M.R.C.P.

The following candidates have been approved at the examinations indicated:

ACADEMIC POSTGRADUATE DIPLOMA IN MEDICAL RADIOLOGY (DIAGNOSIS).—M. M. Craig.

ACADEMIC POSTGRADUATE DIPLOMA IN MEDICAL RADIOLOGY (THERAPY).—H. S. Ali, R. J. Dickson, A. W. Reghers.

Professor A. W. Downie, professor of bacteriology in the University of Liverpool, will deliver a special University Lecture on "Infection and Immunity in Smallpox" at the London School of Hygiene and Tropical Medicine, Keppel Street, Gower Street, London, W.C., on Friday, January 26, at 5.30 p.m.

A course of two special University Lectures on "The Isolation, Properties, and Functions of the Vitamins B_{12}" will be delivered by Dr. E. Lester Smith at the School of Pharmacy, 17, Bloomsbury Square, W.C., on Wednesday and Thursday, February 7 and 8, at 5.30 p.m.

A special University Lecture on "Folic Acid and Growth of Tumours, especially Rous Sarcoma" will be delivered by Professor J. Engelbreth-Holm, professor of pathological anatomy in the University of Copenhagen, at the London School of Hygiene and Tropical Medicine on Monday, February 26, at 5.30 p.m.

All the above lectures are addressed to students of the University and to others interested in the subjects. Admission is free, without ticket.

Medical News

Research on Tuberculosis

The annual report for 1950 of the British Tuberculosis Association's research committee records a small pilot trial using thiosemicarbazone on advanced pulmonary tuberculosis. Although very popular in Germany, this drug does not appear from published reports to be anything like as effective as streptomycin. The trial confirmed these papers to the extent of suggesting that thiosemicarbazone might find its real use as an adjunct with streptomycin when *para*-aminosalicylic acid could not be given. A fuller controlled investigation is planned. The committee is also making a survey among students of London medical schools and in chest clinics to determine whether histoplasmosis is endemic in Britain.

Financial Aid to Private Patients

The Hospital Service Plan, in existence for many years, has been modified to suit present-day conditions. Anyone subscribing to this insurance fund and who is a private ward in-patient may recover the whole cost of hospital and professional fees up to a maximum of £210 in any year. Refunds are also made against the cost of full-time home nursing, and contributors who are general ward N.H.S. patients are also entitled to certain benefits. The annual subscription varies from £2 10s. to 10 guineas, depending on the degree of cover, and full details may be obtained from the London Association for Hospital Services, Tavistock House South, Tavistock Square, London, W.C.1.

Mass Radiography in Eire

According to a report in the *Irish Times*, the Department of Health has just created a limited non-profit-making company, called the National Radiography Association, which will provide a number of miniature mass radiography mobile units to operate throughout Eire wherever the local health authorities desire them and contribute to the finances of the association. It is hoped that eventually all areas will wish to take advantage of the service, which is modelled on that working in Sweden. At the moment mass radiography is already conducted in Dublin and in County Cork.

A Dominion Lecture Tour

At the invitation of the Postgraduate Federations of Australia and of New Zealand Dr. W. N. Pickles, of Aysgarth, Yorkshire, is during the first half of this year giving a series of lectures in these two Dominions. Dr. Pickles will be away from January 18 to June 20.

In Brief

The new president of the Zoological Society of London, in succession to the late Duke of Devonshire, is Field-Marshal Lord Alanbrooke.

Professor T. Ferguson Rodger is chairman, Dr. A. Dick vice-chairman, Dr. Hunter Gillies secretary, and Dr. M. M. Whittet treasurer of the Association of Psychiatrists of the Scottish Western Region for the session 1950–1.

At the annual general meeting of the British Association of Plastic Surgeons on November 24, 1950, Mr. A. B. Wallace (Edinburgh) was elected president, Mr. R. J. V. Battle (London) vice-president, Mr. R. P. Osborne (Liverpool) treasurer, Mr. J. P. Reidy (London) secretary, and Mr. A. B. Wallace editor. The members of the council are Mr. J. N. Barron, Mr. G. M. FitzGibbon, Professor T. P. Kilner, Sir Archibald McIndoe, Mr. J. S. Tough, and Mr. Rainsford Mowlem.

On December 11 the University of Nancy conferred on Dr. Brock Chisholm, Director-General of the World Health Organization, the degree of doctor *honoris causa* of the University and of the Medical Faculty.

The Royal College of Physicians has prepared a list of all the serial publications, including year books, contained in its library, which specializes in the history of medicine.

Wills

Dr. A. G. N. Weatherhead, of Stretford, Lancs, left £11,548; Dr. H. G. Morris, late M.O. to Walsall and West Bromwich districts schools, left £18,731; Dr. W. E. Audland, of Oxford, £24,959; Dr. H. Dodgson, of Shoreham-by-Sea, £24,910; Dr. John Milne, of Sunderland, £14,455; and Dr. John Clarke, late of Rotherham, £24,172.

COMING EVENTS

Robert Campbell Memorial Oration

A Robert Campbell Memorial Oration will be given by Lieutenant-General Sir William MacArthur in the Great Hall of Queen's University, Belfast, on Thursday, January 11, at 8.30 p.m. His subject is "A Medical Survey of the Irish Famine of 1846."

Goulstonian Lectures

Dr. H. J. Anderson will deliver the Goulstonian Lectures before the Royal College of Physicians of London (Pall Mall East, S.W.) on Tuesday and Thursday, January 9 and 11, at 5 p.m. His subject is "Tuberculosis of the Pericardium."

British Postgraduate Medical Federation

The British Postgraduate Medical Federation (2, Gordon Square, London, W.C.) has arranged a number of lectures on "The Scientific Basis of Medicine," comprising the second half of the series which began in October, 1950, to be delivered at the London School of Hygiene and Tropical Medicine, Keppel Street, Gower Street, W.C., on Tuesdays and Thursdays, from January 9 to March 20, at 5.30 p.m. The lectures are designed especially for research workers and specialists in training, and admission is free, without ticket. Details will be published in the "Societies and Lectures" column of the *Journal* week by week.

Psychiatric Aspects of Acromegaly

A public lecture will be delivered by Professor Manfred Bleuler, of the University of Zurich, at the Institute of Psychiatry, Maudsley Hospital, Denmark Hill, London, S.E., on Wednesday, February 14, at 2.45 p.m. His subject is "Acromegaly and Acromegaloid: Psychiatric and Genetic Aspects."

Lettsomian Lectures

Dr. S. Cochrane Shanks will deliver the Lettsomian Lectures before the Medical Society of London (11, Chandos Street, Cavendish Square, W.) on February 19 and 28 and March 5 at 8.30 p.m.

Osler Club of London

The next meeting will be held at the Medical Society of London, 12, Chandos Street, W.1, on Friday, January 19, 1951, at 7.45 for 8 p.m. The meeting will be in honour of Sir James Cantlie, the centenary of whose birth falls on January 17. It will be opened with a biographical account by Lieutenant-General Sir Neil Cantlie, Director-General, Army Medical Services. The discussion will be opened by Sir Philip Manson-Bahr and Mr. E. D. D. Davis.

There will be a meeting on Friday, March 30, 1951, when Dr. Hugh Sinclair will read a paper on the history of nutrition.

Heberden Society

A clinical meeting of the Heberden Society will be held in the Meyerstein Theatre, Westminster Medical School, Horseferry Road, London, S.W., on Wednesday, January 24, at 4 p.m., when many of the cases shown previously will be demonstrated again after one year's progress, together with new cases. Dr. J. H. H. Glyn, for Dr. W. S. C. Copeman and Dr. Oswald Savage, will show a short film on "The Therapeutic Effects of Cortisone."

Nutrition Society

A meeting of the Nutrition Society will be held at the London School of Hygiene and Tropical Medicine, Keppel Street, Gower Street, W.C., on Saturday, January 20, at 10.30 a.m., when there will be a discussion on "The Comparative Merits of Animal and Vegetable Foods in Nutrition." The speakers will be: Dr. Tristram (St. Andrews), Dr. Carpenter (Rowett Institute), Dr. Blaxter (Hannah Dairy Research Institute), Dr. Yates (Rothamsted Institute), Dr. Lucy Wills (Royal Free Hospital), Dr. Harriette Chick (Cambridge), and Dr. Deane (Cambridge).

British Association of Physical Medicine

A two-day meeting of the British Association of Physical Medicine (45, Lincoln's Inn Fields, London, W.C.) will be held on Friday and Saturday, April 20 and 21, with the following provisional programme: April 20, 11.30 a.m., lecture-demonstration on "Micro-waves," by Dr. A. C. Boyle; 2.15 p.m., meeting at Headley Court Rehabilitation Centre, Epsom; 7 for 7.30 p.m., annual dinner at the Royal College of Surgeons of England. April 21, 9.30 a.m., annual general meeting; 10.30 a.m., short papers; 2 p.m., demonstration on "Ultrasonics." Further information may be obtained from the honorary secretary of the association at the above address.

Medico-Legal Society

The annual dinner of the Medico-Legal Society will be held at the Savoy Hotel, Strand, London, W.C., on Thursday, January 11, at 7.15 for 7.45 p.m.

SOCIETIES AND LECTURES

A fee is charged or a ticket is required for attending lectures marked ●. Application should be made first to the institution concerned.

Monday

INSTITUTE OF PSYCHIATRY, Maudsley Hospital, Denmark Hill, London, S.E.—January 8, 5.30 p.m., lecture-demonstration by Dr. E. Stengel.

MEDICAL SOCIETY OF LONDON, 11, Chandos Street, Cavendish Square, London, W.—January 8, 8.30 p.m., discussion: "*Unexpected Death Following Operation*," to be introduced by Drs. Keith Simpson and Ivan Magill.

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—January 8, 4 p.m., "*Optics (Revision)*," by Mr. H. N. Reed.

●ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—January 8, 5.30 p.m., "*Anatomy of the Eye and Orbit*," by Professor T. Nicol.

Tuesday

BRITISH POSTGRADUATE MEDICAL FEDERATION.—At London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C., January 9, 5.30 p.m., "*The Significance of Acetylcholine as a Chemical Transmitter of the Effects of Nerve Impulses*," by Sir Henry Dale, O.M., F.R.S.

CHELSEA CLINICAL SOCIETY.—At South Kensington Hotel, 47, Queens Gate Terrace, London, S.W., January 9, 7 for 7.30 p.m., coloured cinematograph film: "*Danish Exploration in Northern Greenland*," to be demonstrated by Mr. Ebbe Munck.

●INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—January 9, 5 p.m., "*The Pyogenic Cocci in Dermatology*," by Dr. J. O. Oliver.

ROYAL COLLEGE OF PHYSICIANS OF LONDON, Pall Mall East, London, S.W.—January 9, 5 p.m., "*Tuberculosis of the Pericardium*," Goulstonian Lecture by Dr. H. J. Anderson.

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—January 9, 5 p.m., "*Surgery of the Epiphora*," by Mr. T. M. Tyrrell.

Wednesday

●INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—January 10, 5 p.m., "*X-ray Technique*," by Dr. C. W. McKenny.

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—January 10, 5.30 p.m., "*Retrolental Fibroplasia*," by Mr. R. P. Crick.

ROYAL FACULTY OF PHYSICIANS AND SURGEONS OF GLASGOW, 242, St. Vincent Street, Glasgow.—January 10, 5 p.m., "*Symptoms in Cardiac Diagnosis*," Finlayson Memorial Lecture by Sir John Parkinson.

ROYAL MICROSCOPICAL SOCIETY, Tavistock House South, Tavistock Square, London, W.C.—January 10, 6 p.m., meeting of Biological Section. "*Micro-dissection Studies on Amoebae*," by Dr. Joan Lorch

ROYAL SANITARY INSTITUTE, 90, Buckingham Palace Road, London, S.W.—January 10, 2.30 p.m., discussion: "*The Height of Rooms in Dwellings in Relation to Health and Comfort*," to be opened by Professor G. P. Crowden.

SOCIETY OF CHEMICAL INDUSTRY: FOOD GROUP.—At Medical Society of London, 11, Chandos Street, Cavendish Square, W., January 10, 2.15 p.m., joint meeting of the Microbiological Panel with the Society for Applied Bacteriology. Symposium: "*The Cytology of Bacteria*." Papers by Dr. C. Robinow, Mr. E. O. Morris, Dr. V. E. Cosslett, and Dr. K. A. Bisset. Professor R. J. V. Pulvertaft will open the discussion.

Thursday

ALFRED ADLER MEDICAL SOCIETY.—At 11, Chandos Street. London, W., January 11, 8 p.m., "*A General Theory of Human Behaviour*," by Dr. M. G. Good. Discussion to be opened by Dr. James Moore.

BRITISH POSTGRADUATE MEDICAL FEDERATION.—At London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C., January 11, 5.30 p.m., "*Special Problems of Chemical Transmission of Nerve Effects by Acetylcholine, Adrenaline, and Noradrenaline*," by Dr. W. Feldberg, F.R.S.

DONCASTER MEDICAL SOCIETY.—At the Danum Hotel, Doncaster, January 11, 7 for 7.15 p.m., annual dinner and dance to be held in conjunction with the Doncaster Division.

●INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—January 11, 5 p.m., "*Pathology*," by Dr. J. O. Oliver.

INSTITUTE OF LARYNGOLOGY AND OTOLOGY, 330, Gray's Inn Road, London, W.C.—January 11, 5 p.m., "*Otitic Baratrauma*," by Air Commodore E. D. D. Dickson.

LIVERPOOL MEDICAL INSTITUTION, 114, Mount Pleasant, Liverpool.—January 11, 8 p.m., papers: "*Chemotherapy in Tuberculosis*," by Drs. O. F. Thomas, R. M. Todd, and C. A. St.Hill.

PHYSICAL SOCIETY: COLOUR GROUP.—At Lecture Theatre, Institute of Ophthalmology, Judd Street, London, W.C., January 11, 5 p.m., 57th Science Meeting. "*Colour Vision Characteristics of Egyptian Trichromats*," by Dr. I. G. H. Ishak (Ibrahim Pasha University, Cairo).

QUEEN'S UNIVERSITY (GREAT HALL), Belfast.—January 11, 8.30 p.m., "*A Medical Survey of the Irish Famine of 1846*," by Lieutenant-General Sir William MacArthur.

ROYAL ARMY MEDICAL COLLEGE.—At Lecture Theatre, John Islip Street, London, S.W., January 11, 5 p.m., "*The Significance of Anaemia*," by Sir Lionel Whitby.

ROYAL COLLEGE OF PHYSICIANS OF LONDON, Pall Mall East, London, S.W.—January 11, 5 p.m., "*Tuberculosis of the Pericardium*," Goulstonian Lecture by Dr. H. J. Anderson.

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—January 11, 5 p.m., "*Methods of Examination*," by Mr. C. M. Shafto.

ST. GEORGE'S HOSPITAL MEDICAL SCHOOL, Hyde Park Corner. London, S.W.—January 11, 4.30 p.m., lecture-demonstration on psychiatry.

TORQUAY AND DISTRICT MEDICAL SOCIETY.—At Torbay Hospital, January 11, 4.30 p.m., clinical meeting.

Friday

●ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—January 12, 5.30 p.m., "*Physiology of the Eye*."

ROYAL MEDICAL SOCIETY, 7, Melbourne Place, Edinburgh.—January 12, 8 p.m., "*Myocardial Ischaemia*," dissertation by Mr. Philip Kessly.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

May.—On December 17, 1950, at Chatham, Kent, to Dr. Madeleine (formerly MacKenzie), wife of Dr. G. A. (John) May, a son.

MARRIAGES

Cammock—Fawcett.—On December 18, 1950, at Newcastle-upon-Tyne, Douglas William Cammock to Ruth Margaret Fawcett, M.B., B.S., D.C.H.

Hill—Burns.—On November 30, 1950, at the South Parish Church, St. Finbarr, Cork, John Felim Brian Hill, M.B., B.Ch., 347, High Road, Leyton, London, E., to Kathleen Burns, Douglas Street, Cork.

DEATHS

Murray.—On December 15, 1950, at Stratford-on-Avon Hospital, Donald Sutherland Murray, M.D., Old Town Cottage, Stratford-on-Avon, aged 75.

Neil.—On December 17, 1950, George Loudon Neil, O.B.E., L.R.C.P.& S.Ed., L.R.F.P.S.Glas., of Mayfield, Horbury, Wakefield.

Newby.—On December 14, 1950, at 28, Fairfield Road, Croydon, Surrey, Gervase Edward Newby, O.B.E., F.R.C.S., aged 81.

Panton.—On December 27, 1950, at his home, Otter's Pool, Bourne End, Bucks, Sir Noël Panton, M.B., B.Ch.

Vaughan.—On December 13, 1950, at Sully Hospital, Glamorgan, Arthur Christopher Toulmin Vaughan, M.B., Ch.B., 3, Howell's Crescent, Llandaff, aged 41.

White.—On December 17, 1950, at his home, John Simpson White, O.B.E., M.B., B.Ch., 3, Park Place, Molesworth, Plymouth.

EPIDEMIOLOGICAL NOTES

Smallpox at Brighton

Up to January 1 (6 p.m.) 11 cases of smallpox, including one death, have been reported in the region of Brighton. The infection is thought to have been introduced by an R.A.F. officer who flew from Karachi to Leuchars, Fife-shire, landing there on November 27, 1950. He had been vaccinated in October, 1950. Two days later he travelled to Brighton by rail and felt ill during the journey, suffering from rigors. No signs or symptoms characteristic of smallpox developed, though he is said to have had a sparse rash not seen by a doctor, and he was treated at Brighton for malaria. He visited friends at Brighton, returned to Leuchars on December 12, went to Kinloss, Morayshire, on December 14, and then returned to Brighton on December 21.

Meanwhile, on December 12 a woman aged 27, whom he had been visiting, fell ill. She had had conjunctivitis for two days, and went off duty on December 12, thinking that the malaise that started on that day was due to the conjunctivitis. She stayed at home until December 18, when a few spots developed and she was sent to hospital. Small-pox was diagnosed on December 28. The rash was atypical and the symptoms were mild. On December 27 her father, aged 53, was admitted to hospital very ill, with an erythema-tous and haemorrhagic rash; he died on December 29. Father and daughter had been vaccinated only in infancy.

On December 29 two more cases were diagnosed—a woman friend of the daughter, aged about 25, who had helped to nurse her at home, and a man who had been visiting the family. On December 30 one of the nurses at the hospital developed smallpox, and so on the same day did a woman sorter working at a laundry to which the officer's clothes had been sent on December 13. On December 31 three more cases were diagnosed—two women sorters from the same laundry and another nurse at the hospital. On January 1 smallpox was diagnosed in a third nurse. Two of these nurses had never been vaccinated, and the third once only in childhood.

Poliomyelitis

Notifications in the week ending December 23, 1950, were: paralytic 38 (82); non-paralytic, 15 (25); total, 53 (107). The figures in parentheses are those for the previous week. Uncorrected notifications for the corresponding week in 1947 and 1949 were 60 and 81 respectively. Total un-corrected notifications for 1950 to and including the week under review are 8,643. The corresponding figures for 1947, 1948, and 1949 were 9,147, 2,356, and 6,791 respectively. County areas with the largest number of notifications were London 6 and Essex 6. The marked fall in the figures, equivalent to a 50% reduction compared with the previous week, is very satisfactory.

Influenza

There has been a marked prevalence of influenza in Scandinavia, from cases of which a virus has been isolated not unlike the "A prime" strain which occurred in this country in 1949. The disease has been of a mild type but with a high attack rate. High incidences of influenza have also been reported within recent weeks from certain parts of the U.S.A., Northern Spain, Sardinia, Persia, and elsewhere.

In the absence of notification of influenza, the incidence of pneumonia cases and deaths from pneumonia and influenza are used as a guide to the prevalence of the disease, and the following figures may be of interest.

Great Towns	Pneumonia Including Primary Influenza		
	Cases	Deaths	Influenzal Deaths
Week ending December 9 ..	347	236	34
Week ending December 16 ..	274	283	33
Week ending December 23 ..	363	350	54

England and Wales	Pneumonia (Primary and Influenzal) Cases
Week ending December 9	573
Week ending December 16	684
Week ending December 23	647

Cities and towns with the largest number of pneumonia notifications for the week ending December 23 were: London, 42; Sunderland, 24; Newcastle, 23; Birmingham, 20; Manchester, 17; Sheffield, 15; Liverpool, 14.

Although these statistics afford no evidence of any serious outbreak of influenza in this country up to December 23, in the last week or more the disease has been very prevalent in the north-east, and a number of large towns have been hit, including Sunderland, Durham, and Newcastle-upon-Tyne. It seems likely that the disease has spread to this area from Scandinavia, and laboratory studies may show the virus strain to be identical with that in Scandinavia.

The disease in the north-east appears to be distinct from the so-called influenza at present widespread in other parts of the country. The infection may spread southwards in the next few weeks.

Road Accidents

According to the Ministry of Transport, 483 people were killed in road accidents in October, 1950, as compared with 380 in October, 1949. Those injured in road accidents in October, 1950, numbered 4,346 seriously injured and 12,870 slightly injured. Corresponding figures for October, 1949, are 3,738 and 11,514. October, 1950, had the highest number of road casualties for any October since the war. In November, 1950, the provisional figure for deaths is 484.

Week Ending December 16

For eight consecutive weeks the incidence of dysentery has risen in England and Wales, and during the week end-ing December 16 a new record was established with 1,214 notifications. A further rise in the number of notifications of measles and whooping-cough was recorded, and both these diseases are at an exceptionally high level for this period of the year. A rise was recorded in the incidence of scarlet fever and acute pneumonia, but the seasonal trends of these diseases are unusually low. Of the 50 cases of diphtheria in England and Wales, 14 were notified in Lancashire.

The fall in the notifications of acute poliomyelitis was checked; a rise of 3 was recorded, and the total was the largest number notified during any fiftieth week. The largest of the local rises of acute poliomyelitis during the week were 5 in Surrey and 4 in Kent; in the latter county 8 of the 10 cases were notified in Orpington U.D.

Dysentery

The largest returns of dysentery during the week in England and Wales were: Lancashire 262 (Southport C.B. 52, Oldham C.B. 28, Rishton U.D. 28, Irlam U.D. 26, Salford C.B. 23, Great Harwood U.D. 21, Liverpool C.B. 20, Middleton M.B. 16); Leicestershire 239 (Leicester C.B. 196, Market Harborough R.D. 13, Barrow-upon-Soar R.D. 10); Yorkshire West Riding 132 (Earby U.D. 48, Saddle-worth U.D. 20, Leeds C.B. 10, Sheffield C.B. 10, Bradford C.B. 9); Shropshire 53 (Shrewsbury M.B. 20, Whitchurch U.D. 10); London 52 (Islington 14, St. Pancras 11); Surrey 47 (Malden and Coombe M.B. 20, Esher U.D. 18); Durham 40 (Darlington C.B. 14); Middlesex 40 (Edmonton M.B. 29); Monmouthshire 40 (Abercarn U.D. 20, Newport C.B. 15); Warwickshire 39 (Birmingham C.B. 33); Yorkshire East Riding 37 (Kingston-upon-Hull C.B. 34); Cheshire 34 (Macclesfield M.B. 9); Staffordshire 25 (Cheadle R.D. 9); Yorkshire North Riding 23 (Middlesbrough C.B. 15); Northamptonshire 19 (Corby U.D. 13); Essex 15 (Rom-ford M.B. 12); Wiltshire 14 (Marlborough and Ramsbury R.D. 8); Cumberland 12; Glamorganshire 12; Gloucester-shire 11.

No. 50

INFECTIOUS DISEASES AND VITAL STATISTICS

We print below a Summary of Infectious Diseases and Vital Statistics in the British Isles during the week ended December 16.

Figures of Principal Notifiable Diseases for the week and those for the corresponding week last year, for: (a) England and Wales (London included. (b) London (administrative county). (c) Scotland. (d) Eire. (e) Northern Ireland.

Figures of Births and Deaths, and of Deaths recorded under each Infectious Disease, are for: (a) The 126 great towns in England and Wales (including London). (b) London (administrative county). (c) The 16 principal towns in Scotland. (d) The 13 principal towns in Eire. (e) The 10 principal towns in Northern Ireland.

A dash — denotes no cases; a blank space denotes disease not notifiable or no return available.

Disease	1950					1949 (Corresponding Week)				
	(a)	(b)	(c)	(d)	(e)	(a)	(b)	(c)	(d)	(e)
Meningococcal infection	29	—	14	1	1	37	2	17	3	1
Deaths		—	—				—	2		
Diphtheria	50	4	11	2	2	92	14	31	5	4
Deaths	—	—	—	—	—	1	—	—	—	—
Dysentery	1,214	52	147		7	197	13	75	—	2
Deaths		—			—				—	—
Encephalitis, acute	6	1	1		—	—	—	—	1	—
Deaths		—		—			—			
Erysipelas			21	10	4			38	10	4
Deaths							—			
Infective enteritis or diarrhoea under 2 years				18	10				18	
Deaths	26		3	1	2	23	1	5	3	
Measles*	14,223	1534	255	157	100	1,793	34	37	57	156
Deaths†		—	—	2	—			—	—	—
Ophthalmia neonatorum	23	—	12		1	31	—	8	—	—
Deaths										
Paratyphoid fever	5	—	3(B)		—	8	1	—	—	—
Deaths	—		—		—	—	—	—	—	—
Pneumonia, influenzal	684	36	19		10	676	38	15	4	14
Deaths (from influenza)‡	33	2	6	—	—	41	5	—	—	—
Pneumonia, primary			272	¶				390	33	
Deaths	283	38		7	21	252	40		14	11
Poliomyelitis, acute	107	5	6	4	1	93	11	1	1	1
Deaths§	8	2		—		6	1			
Puerperal fever			7				—	5		
Deaths										
Puerperal pyrexia‖	66	3	9		—	73	6	3	1	3
Deaths							—			
Relapsing fever	—	—			—	—	—			—
Deaths										
Scarlet fever	1,348	101	203	82	37	2,829	190	245	89	226
Deaths†		—	—	—	—			—	—	—
Smallpox	—	—	—		—	—	—	—	—	—
Deaths					—				—	—
Typhoid fever	2	—	—	—	—	5	—	—	2	—
Deaths	—	—	—		—	—	—	—	—	—
Typhus fever	—	—	—	—	—	—	—	—	—	—
Deaths					—				—	—
Whooping-cough	4,503	424	620	68	35	984	32	66	26	58
Deaths	9	1	—	—	—	—	—	—	—	—
Deaths (0–1 year)	258	27	37	21	17	281	30	44	20	11
Deaths (excluding stillbirths)	6,080	924	789	234	172	5,607	838	699	187	144
Annual death rate (per 1,000 persons living)			15·9					14·0	11·6	
Live births	6,436	1092	823	261	190	6,711	1066	861	244	196
Annual rate per 1,000 persons living			16·5					17·3	15·1	
Stillbirths	178	22	28			189	25	23		
Rate per 1,000 total births (including stillborn)			33					26		

* Measles is not notifiable in Scotland, and the returns are therefore an approximation only.

† Deaths from measles and scarlet fever for England and Wales, London (administrative county), will no longer be published.

‡ Includes primary form for England and Wales, London (administrative county), and Northern Ireland.

§ The number of deaths from poliomyelitis and polio-encephalitis for England and Wales, London (administrative county), are combined.

‖ Includes puerperal fever for England and Wales and Eire

¶ Primary pneumonia no longer notifiable in Eire.

Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Alcohol after Head Injury

Q.—*How soon is it advisable to resume moderate drinking after head injury?*

A.—Drinking may be harmful to a patient who has had a head injury, in two ways: (1) In cases of open skull fracture with penetration of the cerebrum post-traumatic epilepsy may appear as a complication. If it does, heavy consumption of beer, by causing hydration, may precipitate fits. (2) A patient who has sustained a severe and diffuse injury to the brain, followed by a long period of unconsciousness, will often exhibit such symptoms as disturbance of memory, power of concentration, or mood. In the presence of such symptoms drinking of spirits is best avoided. Finally, anything likely to produce headache in a patient with this symptom should be avoided. This in itself indicates care in the matter of alcoholic intake.

Treatment of Malignant Hypertension

Q.—*A man of 55, with long-standing hypertension (B.P. 246/150), had a stroke three months ago from which he recovered; one month ago he awoke with a paresis of the left leg which rapidly improved, but 12 hours later he suddenly lost the sight of both eyes. Examination showed pallor of the retinae and constriction of the arteries, and there was loss of peripheral vision; objects and colours could, however, be distinguished with difficulty. Now (one month later) there is slight improvement in vision, the retinae are less pale, and the disks appear normal. What are the diagnosis, treatment, and prognosis? Would hexamethonium bromide or venesection be of value in this case?*

A.—This man appears to be suffering from malignant hypertension with episodes of severe vascular spasm. He should improve with rest in bed, a low-sodium diet, and hexamethonium bromide. Venesection should not be undertaken, because it would do no lasting good and might well prove dangerous. In these cases the patient should be thoroughly examined first, particularly with regard to cardiac and renal function. The effect of sedation should then be ascertained. Hexamethonium bromide may then be administered subcutaneously or intramuscularly in an initial dose of 20 mg., preferably with the patient in the propped-up position in bed. A fall in blood pressure may cause dizziness, but this can be promptly overcome by removing one or two pillows or, if necessary, by laying the patient flat. The second dose may be increased or reduced according to the initial response. Injections may then be given twice daily. As soon as satisfactory control is obtained, hexamethonium bromide may be given by mouth up to a maximum of about 1 g. three times a day.

Note.—In reply to a question on the immediate treatment of an attack of hypertensive encephalopathy ("Any Questions?", November 25, 1950, p. 1235) the intravenous administration, over a period of two minutes, of pentamethonium iodide or hexamethonium iodide in doses of 30–40 mg. was recommended. The injection should be given with the patient propped up; the onset of dizziness is a signal to stop the injection and to lay the patient flat, because this means that a satisfactory fall of blood pressure has been obtained and the full dose is unnecessary and may provoke too severe a reaction. The hexamethonium compounds should not be given lightly in general practice, their successful administration requiring considerable knowledge and experience.

Intrapleural P.A.S.

Q.—*Has intrapleural para-aminosalicylic acid (P.A.S.) been tried in the treatment of pulmonary tuberculosis? If so, with what results?*

A.—Solutions of *para*-aminosalicylic acid have been used extensively in the treatment of tuberculous empyemata. P.A.S. is given intrapleurally usually in the form of a 20% solution of the sodium salt, 20 to 40 ml. being injected weekly, according to the severity of the case. The results are variable and depend on the degree of thickening of the pleura and the chronicity of the lesion. The presence of a bronchopleural fistula, although not an absolute contra-indication, will have a serious adverse effect on the treatment.

To obtain the best results all pus must be aspirated from the pleural cavity, and the cavity washed out with normal saline, before the injection of the P.A.S. solution. In cases with secondary infection this should be treated with penicillin before using P.A.S. Local anaesthetics containing *para*-aminobenzoic acid should not be used with P.A.S., as *para*-aminobenzoic acid antagonizes P.A.S.

In this country the most favourable results have been recorded by T. G. Dempsey and M. H. Logg (*Lancet*, 1947, **2**, 871; *Tubercle*, 1948, **29**, 216).

V.D. Prophylaxis

Q.—*What are the best measures of personal prophylaxis against venereal disease?*

A.—A condom, carefully put on and carefully removed, will almost certainly prevent infection of the parts covered, but not, of course, of the surrounding skin.

Thorough washing of the whole genital area with soap and water, followed by the application of calomel cream (containing a sulphonamide or mercury oxycyanide), will prevent infection in the vast majority of cases if carried out immediately after exposure.

Penicillin tablets (300,000 units) swallowed within a few hours of exposure are said to prevent infection in a high proportion of cases.

Sulphadiazine, one or two grammes taken at once followed by a similar dose next morning, is likely to prevent the development of gonorrhoea but not of syphilis.

A few capsules of "aureomycin," each of 250 mg., swallowed soon after exposure will probably prevent infection with either gonorrhoea or syphilis, but few investigations with this antibiotic have yet been reported.

It will be clear from the foregoing that the sooner and more thoroughly prophylaxis is applied the greater will be the chances of preventing the development of infection.

Amphetamine and Cardiac Arrhythmia

Q.—*Is amphetamine likely to lead to auricular fibrillation in a healthy person, or in a person who suffers from mitral stenosis of rheumatic origin and has a history of several bouts of auricular fibrillation?*

A.—Amphetamine is unlikely to lead to auricular fibrillation in a healthy person. But on the whole it is wise not to use such drugs for patients who have a tendency to cardiac arrhythmias, and certainly not for one who suffers from mitral stenosis and who has had several bouts of paroxysmal auricular fibrillation.

Lupus Erythematosus

Q.—*What is known of the aetiology of lupus erythematosus? What is the treatment of the acute disseminated and the chronic localized varieties respectively?*

A.—Chronic and acute lupus erythematosus are thought by most authorities to be related, and occasionally the one is seen to pass into the other, though more often the types retain their identity throughout their course. Their aetiology is unknown, but is associated with a disturbance of vascular function and sometimes to the adjustment of this to climatic conditions. Focal sepsis, drug therapy, and sometimes other disorders may act as predisposing factors.

Empirical treatment with gold injections is at present the most helpful measure in the management of the chronic form of lupus erythematosus.

In the acute type, adrenocorticotropic hormone (A.C.T.H.) and cortisone produce dramatic results, but the disease returns as badly as ever on their withdrawal. Some cases have now been reported in which prolonged administration of these substances has maintained improvement. In some cases this improvement has lasted for periods of months after the cessation of therapy.

Chronic Otitis Externa

Q.—*A girl of 21 has had bilateral chronic otitis externa for 18 months. The condition began as a series of boils in the external meatus, and has proved resistant to orthodox measures. What treatment is suggested?*

A.—It would be worth trying irradiation, either by *x* rays, the appropriate dose being determined by a radiologist, or by the local application of an alcoholic solution or varnish containing thorium X.

Light-headedness in Elderly Diabetic

Q.—*A bachelor, aged 72, with a mild degree of diabetes mellitus controlled by diet, complains of a sensation of light-headedness immediately after evacuation of the bowels every morning after breakfast. This persists for two or three hours. His blood pressure is only slightly raised. He also suffers from angina of effort. What is the cause of this sensation, and is there any treatment?*

A.—It seems probable that this symptom is related to transient disturbance of the cerebral circulation, particularly as the presence of angina indicates arteriosclerosis. It is advisable to ensure that defaecation should be as nearly effortless as possible, and to this end liquid paraffin should be prescribed to keep the intestinal contents soft. If possible the patient should breakfast before he has dressed, and, after the bowels have been evacuated, he should return to bed for 15–30 minutes. On first waking he should take ½ gr. (32 mg.) of phenobarbitone.

Sterilization of Syringes

Q.—*What antiseptic miscible with oil could be recommended for adding to the lubricant of an injection syringe? What concentration would be necessary to guarantee the sterility of the syringe?*

A.—It is impossible to sterilize a syringe by this method. Even germicides in solution in water do not penetrate the crevices, and all chemical methods of sterilizing syringes have been found ineffective. Obviously areas not reached by an aqueous solution would be even more inaccessible to a viscous oil. Lubrication and sterilization (by dry heat or autoclaving) should be separate processes. Full instructions, and substantiation of the statements made, will be found in *The Sterilization, Use and Care of Syringes* (Medical Research Council War Memorandum No. 15, H.M. Stationery Office, 1945).

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